CHAPTER 5

A COMPARISON OF ALTERNATIVE SOURCES OF AIR QUALITY DATA

1. Introduction

     As previously indicated, information from the "St. Louis' Pollution -
Morbidity" study, performed by Geomet Technologies, Inc., was to have been
collected concurrently with aerometric data generated by the Regional Air
Monitoring System (RAMS). However, this planned matching of extensive
health and daily activity information with detailed air quality
measurements did not occur when delays in approval of the survey design
prevented implementation of the first phase of data collection until June,
1978. The RAMS system had entirely shut down by March 31, 1977. One of
the reasons for continuing with the Geomet study in spite of this problem
was the existence of locally operated pollution monitoring networks in the
study area including those administered by the city and county of St.
Louis, and the Illinois EPA. The purpose of this chapter is to evaluate
the quality of the aerometric data produced by these three sources. The
results of this evaluation, which are contained in Section 2, suggest that
the three non-RAMS data sources are quite unreliable and should not be used
in an econometric health epidemiology study. Section 3, then, discusses
the constraints placed by this finding on the use of the St. Louis health
data set.

2. Quality of the Non-RAMS Aerometric Data

     Aerometric monitoring systems generally are plagued with measurement
drift problems if highly sensitive devices are used. Correspondingly, if
less sensitive measurement devices are used, changes in ambient pollutant
concentrations may not be accurately detected. The audit documentation
presented in Chapter 4, together with informal opinion related by scholars
in the atmospheric science field indicated that the RAMS was distinguished
by the use of sensitive instruments, the attention paid to the calibration
of those instruments in particular, and overall quality control in general.
Most monitoring devices measuring gaseous pollutants were automatically
recalibrated daily as part of many routine on-site physical inspections and
maintenance procedures. From that evidence, the conclusion was drawn that
the RAMS network data could be taken as the best available benchmark for
measuring actual pollutant levels. Decisions concerning how to best use
the St. Louis health survey data in a particular study design hinge in
large part on the quality of the data from non-RAMS, locally administered
pollution monitoring systems. As a step in judging this quality, a
correlation study was performed which compared the locally-administered

stations' pollutant time series with the corresponding time series from RAMS Stations.

As a first step, pollution data from the appropriate regional EPA administrations were collected for all county, city and Illinois EPA monitoring stations in the St. Louis Air Quality Control Region (AQCR) for the decade 1970-1980, Preliminary analysis of these sources revealed a relatively small degree of overlap between operation dates and a large incidence of missing data for entire days and/or fractions of days. Based upon this preliminary analysis, a procedure was devised for a RAMS vs. non-RAMS pollutant time series comparison. The best quality RAMS data, after making the system operational, were in the latter portion of the system's operation. This distinct improvement in RAMS system quality is a conclusion based both on the audits performed on the RAMS system[1] and discussion with atmospheric scientists familiar with the system. Accordingly, the dates of 1/1/76 - 3/14/77 were chosen as the dates for generating the RAMS benchmark time series, by pollutant. Within these dates, daily averages were computed for the hours of 0600-2000, inclusive. This time frame was chosen because of the importance of activity in exascerbating the negative effects of pollution exposure, the generally higher pollution levels during the day, and because RAMS automatic calibration procedures were performed between the hours of 2000-2400 daily. For each pollutant and station, two time series were generated: the daily means over all valid hourly readings and daily means over the 90 percent decile determined by the daily peak.

Given these benchmark time series, the next step was to generate a time series that matched pollution measurements from non-RAMS and RAMS stations for the purpose of calculating a set of Pearson correlation coefficients. Because of the variation in operation periods and valid data days, the analysis proceeded on a non-RAMS station-pollutant basis. In other words, given a non-RAMS station that operated at least intermittently over the period 1/1/76 and 3/14/77, a data matrix was constructed in order to generate a set of pollutant specific correlations between the non-RAMS station chosen and each of the RAMS stations in the St. Louis area. Between station correlation coefficients were calculated for the following representatives of the major pollutant groups; ozone, sulfur dioxide, nitric oxide, and carbon monoxide.

The actual program generating the time series data matrix by station for a given pollutant proceeded as follows. A date was selected in the non-RAMS time series. If more than twelve hours were missing from that date's hourly measurements, that date was ruled invalid. If a date had measurements for at least the minimum number of valid hours then an average reading over the hours of 0600-2000 was computed. In order to be identical to the RAMS daily average, readings below a threshold of .0025 ppm were classified as missing and did not enter into the average computed for all pollutants except carbon monoxide. If all hours were classified as missing, the date was ruled invalid. When a valid date had been found and a valid average computed, the matching date from the 439-day RAMS time series was read and the nineteen RAMS daily averages and the one non-RAMS daily average were output on an external file. After repeating this

procedure over all candidate dates in the non-RAMS time series, the matrix of valid dates by stations was then input into a standard SPSS Pearson zero-order correlation procedure which generated the correlation coefficients, number of days in the generated time series, and the means and standard deviation of measurements at each station over the time series. Thus, correlations were run between a given non-RAMS station, in turn, with all RAMS stations over identical days with identically-computed daily averages over all readings above a given threshold and over all readings in the daily 90 percent decile.

Results from this data manipulation and analysis are presented in Tables 1-8. St. Louis County operated stations have a CO prefix, St. Louis City stations a CI prefix, and Illinois EPA stations an IL prefix in these tables. The overlays in Figures 1 and 2 compare the location of the stations in each of the four monitoring systems.

For each of the four pollutants there are two tables, one for daily means and one for 90 percent decile averages. The tables of daily means give the correlations of each non-RAMS station with the two or three closest RAMS stations, as well as the correlations between these RAMS stations. In all tables, n.a., indicates that data were not available for a particular pollutant or a particular RAMS station. The tables of daily means also include the mean readings for all stations explicitly considered; the high correlation or correlations among the set of all RAMS stations with the non-RAMS station, and the number of days in the time series generating the above data. The tables showing the 90 percent decile averages do not include correlations between the two or three closest RAMS stations. Each table also contains the critical value of the correlation **coefficient, ρ_c, to test** the null hypothesis $\rho = 0$ using a two-tailed test **at the 5 percent level of** significance. Assuming that the daily mean and 90 percent decile values are normally distributed, then

$$\hat{\rho}[(n - 2)/(1 - \hat{\rho}^2)]^{\frac{1}{2}} \sim t(n - 2) \tag{1}$$

The number of observations used in computing the values for $\hat{\rho}$ exceeded 40 in all cases but two. Consequently, in that overwhelming majority of cases, values for $\hat{\rho}_c$ were computed by substituting the number 2 as an approximation to **the** critical value $t_c(n - 2)$ into equation (2). ρ_c then was computed as

$$\hat{\rho}_c \sim t_c(n - 2)/(n + 2)^{\frac{1}{2}} \tag{2}$$

In both of the two cases where n < 40, n = 16 and the value $t_c(n - 2) = 2.145$ was used.

Tables 1 and 2 present the results for ozone daily means and 90 percent decile averages, respectively. Among the county and Illinois EPA stations and their spatially close RAMS counterparts, the means for both daily means and 90 percent decile measurements agree quite closely except for station CO6 in Table 1. Among the city stations, however, the average daily means are considerably less than the average among close RAMS

stations, although the 90 percent decile means agree fairly closely. An important pattern to notice, however, is the consistently lower correlations between each non-RAMS station and close RAMS stations as compared to correlations between these RAMS stations. In Table 1, the correlations between non-RAMS and RAMS stations range from .10 to .57, while correlations between RAMS stations range from .44 to .88 for daily means. Given the RAMS data as a benchmark of quality, a distinct lack of quality in the non-RAMS systems would be indicated. Particularly striking are the low correlations between non-Rams and RAMS stations which were essentially operating at the same location. For example, the correlations between the Illinois EPA Station IL7 and RAMS Station 104 are -36 for daily means and .31 for 90 percent decile averages. City Station CI5 was located very close to RAMS Station 101 in downtown St. Louis. Their daily means correlation is -15 while their 90 percent decile correlation is .25. In the county system, Station CO4 and RAMS Station 112 are in close proximity. Their daily means correlation is .26 while the 90 percent decile average correlation is .27. In all six of these cases, $\hat{\rho} > \hat{\rho}_c$; thus the null hypothesis $\rho = 0$ should be rejected in favor of the alternative hypothesis, $\rho \neq 0$. However, even though there appears to be some linear association between the ozone readings from adjacent stations, that association simply is too weak to support the use of the non-RAMS in this study. This conclusion obviously is based upon an implicit (and arbitrary) lower bound of "acceptability" for the values of ρ. However that lower bound for adjacent stations surely must exceed .40. In fact, that correlation implies that a linear regression of the RAMS ozone readings on non-RAMS ozone readings would produce an $R^2 = .16$!

Moreover, another anomaly seen in Tables 1 and 2 is the high incidence of highest correlation between a non-RAMS station and the set of nineteen RAMS stations being with RAMS Station 102. Stations as geographically disparate as C06, IL2, and IL6 all were most highly correlated with Station 102 in the 90 percent decile average time series. Although no reasons are given here for this phenomena, it is suggestive of the problems involved in using the non-RAMS data as a source for exposure data. There appear to be anomalous patterns which need to be explained before use of the data can be justified.

As inconsistent as the ozone RAMS and non-RAMS data appear, the other three pollutants checked showed dramatically lower correlations. Tables 3 and 4 display correlations for nitric oxide (NO) computed from daily means and daily 90 percent decile averages, respectively. The daily mean correlations between RAMS and non-RAMS readings range from -.0034 to .21, while the RAMS intercorrelations range from .35 to .76. For the daily 90 percent decile correlations, the RAMS--non-RAMS values ranged from -.04 to .26, with the latter figure being something of an outlier. Choosing stations in close proximity again does not appear to increase the correlation between the NO readings from RAMS and non-RAMS stations. Between Stations IL6 and 109 there is essentially nothing but a random relationship between the two daily means NO time series; indicated by a correlation coefficient of -.007. Between Stations CO4 and 112, the NO daily means time series comparison shows a correlation of .026. For the city system, the same daily mean time series comparison between Station CI5

and 101 generates a correlation of .02. Furthermore, Tables 3 and 4 again
show the anomalous pattern of highest correlation being with RAMS stations
quite far away geographically combined with low correlation with RAMS
stations in close proximity.

Tables 5 through 8 continue to point this same dismal picture of data
quality from the non-RAMS systems. Correlations reported for daily means
and 90 percent decile readings for the pollutants, carbon monoxide and
sulfur dioxide frequently are negative and seldom exceed the critical
values shown for $\hat{\rho}_c$; a situation that persists even when immediately
adjacent stations are considered. For example, the correlations for carbon
monoxide readings on both daily means and daily 90 percent deciles are
negative for IL7 and 104, CI5 and 101, and IL6 and 109. However, the
correlation between daily mean carbon monoxide readings at CO4 and 112 was
.15, which was just slightly greater than the critical value of .1421.
Finally, with respect to sulfur dioxide, there was only one case of
immediately adjacent monitoring station. The correlations between daily
mean and daily 90 percent decile readings for IL7 and 104 were .06 and
-.003 respectively.

3.   <u>Constraints on Using the St. Louis Health Data</u>

Among the four pollutants examined, only the ozone data obtained from
the non-RAMS monitoring networks appears to bear a significant positive
association with concurrent ozone readings from the RAMS network. However,
that association is weak; for immediately adjacent stations it was never
higher than .36. Telephone interviews with officials in charge of
operating the St. Louis City and St. Louis County monitoring systems
reinforced a suspicion as to the likely cause of the poor-quality data:
the lack of a full-scale calibration and maintenance for the instruments in
these systems when the time series were generated.

In any case, because of these rather extraordinary problems with the
non-RAMS sources of air quality data, the full potential of the health
survey data cannot be realized. More specifically, the diary data simply
are unusable in a study designed to assess the consequences of air quality
changes on short-term or acute illness. Such a study would require
reasonably accurate air quality measures matched on a day-by-day basis to
the diary health data. If those air quality measures do not exist, there
would appear to little that can be done to salvage the situation,
regardless of the care that may have been used in collecting the health
data. Of course, the correlation study just reported covered the period
1/1/76-3/14/77 whereas the health survey was conducted between June, 1978
and July, 1979. However, there is no evidence that improved monitoring
procedures were instituted by the non-RAMS systems in the interim. As a
consequence, the empirical work reported in Chapter 8 makes no use of the
health diary information at all. Only data obtained from the Household
Background Questionnaire, the Individual Background Interview, and the
Supplemental Interview were examined. Prior to use, they were matched with
air quality data from the RAMS using a procedure described more fully in
Chapter 6. The RAMS data, then, are interpreted as reflecting historical
or long-term exposure patterns faced by residents of the St. Louis area.
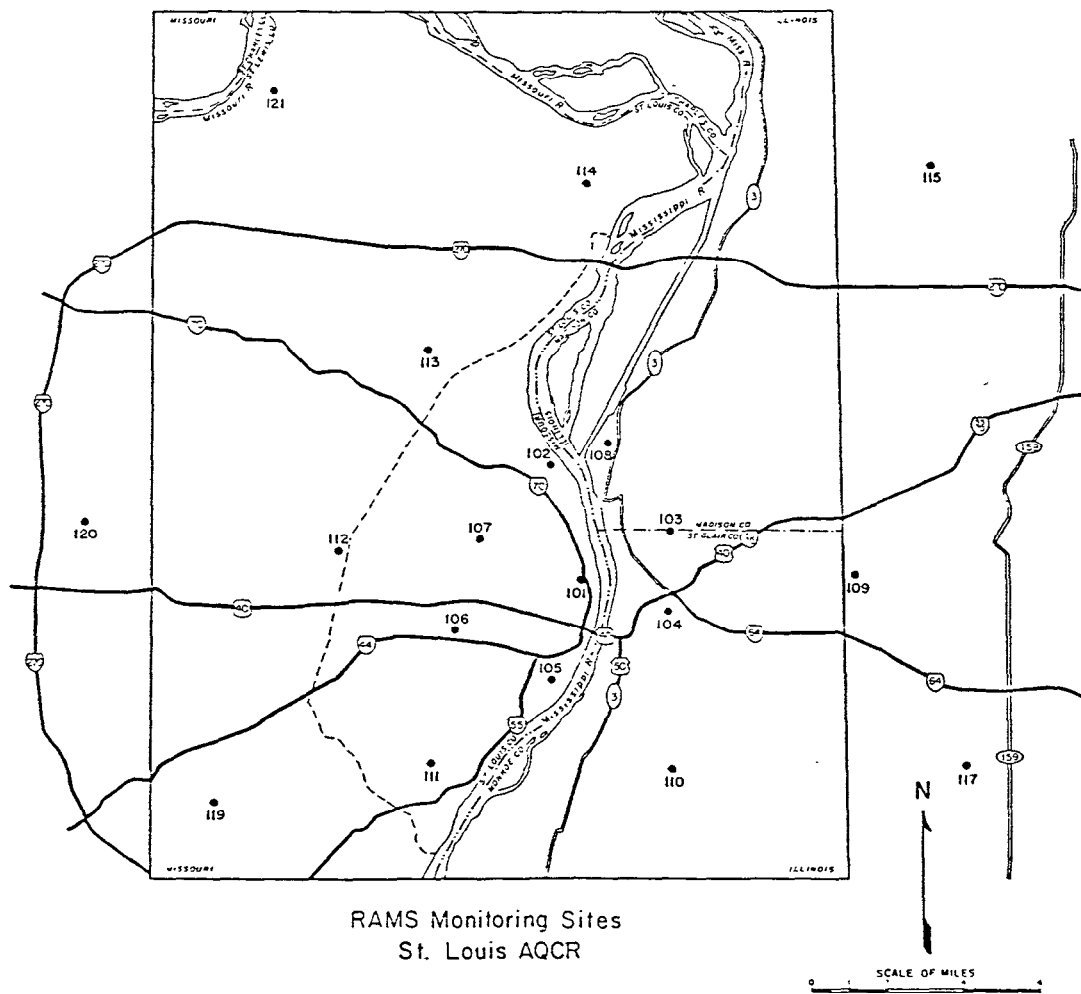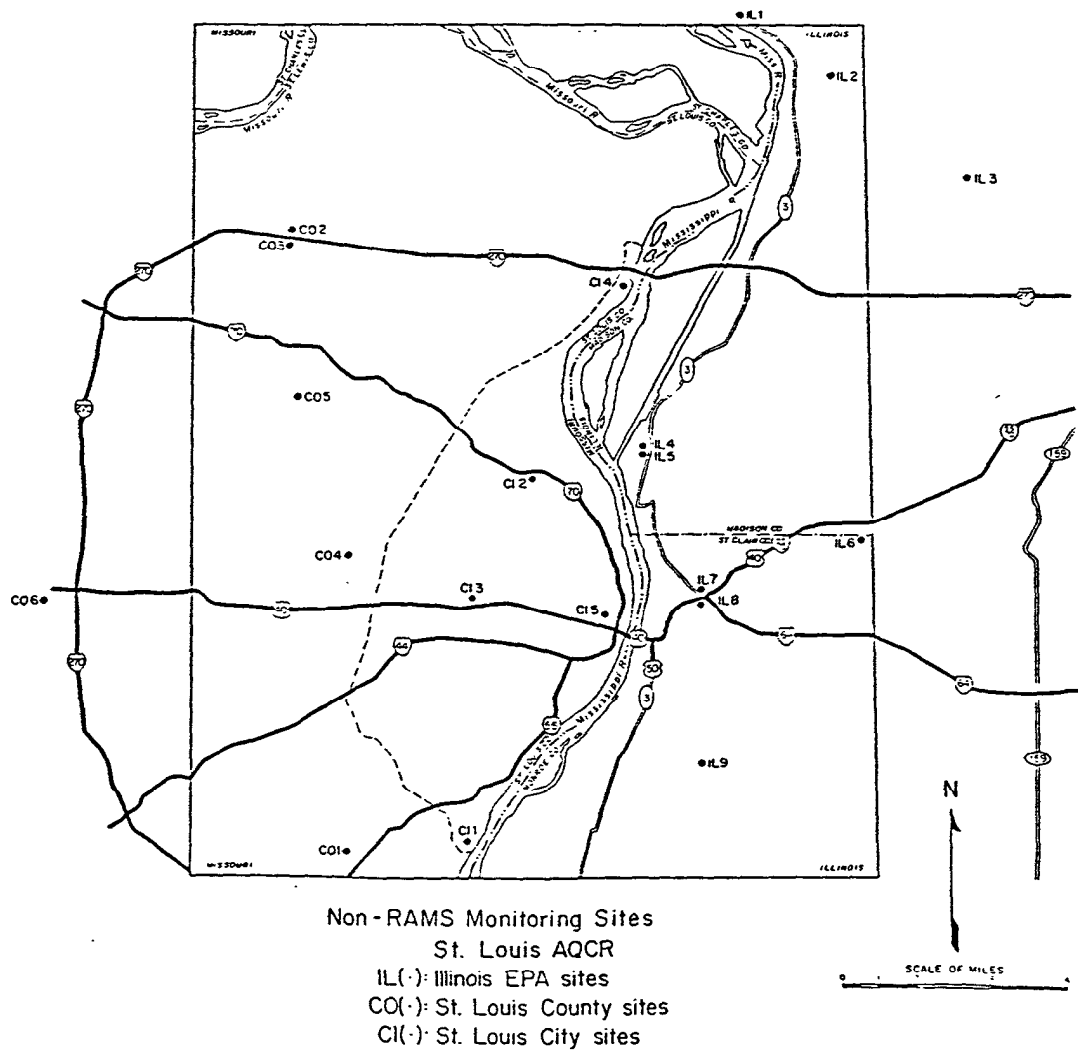
RAMS Monitoring Sites
St. Louis AQCR

Figure I

Non-RAMS Monitoring Sites
St. Louis AQCR
IL(·): Illinois EPA sites
CO(·): St. Louis County sites
CI(·): St. Louis City sites

Figure 2

TABLE 1

CORRELATIONS BETWEEN DAILY MEANS: OZONE

	119	111	110	CO1
CO1 (30001)	.35	.39	.33	
119		.74	.68	
111			.83	
Means:	.026	.023	.029	.028
High:	.453, with 108			
Days:	104			
$\hat{\rho}_c$:	.1943			

	113	120	121	CO2
CO2 (20002)	.42	.28	.44	
113		.68	.79	
120			.62	
Means:	.030	.028	.035	.027
High:	.494, with 111			
Days:	118			
$\hat{\rho}_c$:	.1826			

	112	120	CO4
CO4 (1040001)	.26	.12	
112		.59	
Means:	.023	.023	.026
High:	.419, with 102		
Days:	221		
$\hat{\rho}_c$:	.1339		

	104	103	105	IL7
IL7 (2120008)	.36	.33	.40	
104		.54	.55	
103			.59	
Means:	.018	.021	.020	.017
High:	.535, with 102			
Days:	398			
$\hat{\rho}_c$:	.1000			

(Table 1, Continued)

	109	103	104	
IL6 (2120009)	.26	.20	.27	
109		.64	.55	
103			.54	
				IL6
Means:	.031	.020	.018	.0015
High:	.386, with 102			
Days:	397			
$\hat{\rho}_c$:	.1001			

	112	120	113	
C05 (4120001)	.17	.10	.29	
112		.59	.72	
120			.68	
				C05
Means:	.024	.024	.027	.027
High:	.33, with 102			
Days:	159			
$\hat{\rho}_c$:	.1579			

	111	105	106	
CI1 (4280007)	.27	.20	.19	
111		.71	.77	
105			.66	
				CI1
Means:	.023	.019	.021	.024
High:	.369, with 103			
Days:	214			
$\hat{\rho}_c$:	.1361			

	102	107	113	
CI2 (4280061)	.39	.30	.36	
102		.73	.88	
109			.67	
				CI2
Means:	.024	.019	.025	.003
High:	.39, with 102			
Days:	336			
$\hat{\rho}_c$:	.1088			

(Table 1, Continued)

51

	106	111	112	CI 3
CI 3 (4280062)	.35	.36	.26	
106		.77	.65	
111			.76	
Means:	.025	.027	.025	.003
High:	.41, with 102			
Days:	297			
$\hat{\rho}_c$:	.1157			

	113	114	102	CI 4
CI 4 (4280063)	.47	.42	.57	
113		.80	.87	
114			.82	
Means:	.019	.026	.018	.003
High:	.57 with 102			
Days:	249			
$\hat{\rho}_c$:	.1262			

	101	105	106	CI 5
CI 5 (4280064)	.15	.16	.17	
101		.65	.73	
105			.66	
Means:	.026	.022	.025	.002
High:	.26 with 102			
Days :	294			
$\hat{\rho}_c$:	.1162			

	120	119		C06
C06 (4300006)	.22	.46		
120		.54		
Means :	.024	.027		.0025
Highs:	.62, with 102.			
Days :	226			
$\hat{\rho}_c$:	.1325			

(Table 1, Continued)

		115	114	121	
IL2 (8520007)		.31	.46	.45	
115			.46	.44	
114				.74	
					IL2
Means:		.026	.031	.031	.0025
Highs:	.57, with 102				
Days:	402				
$\hat{\rho}_c$:	.0995				

Table 2, continued

	101	105	106	
CI5 (4260064)	.25	.24	.21	
				CI5
Means:	.046	.036	.042	.028
High:	.31 with 102			
Days:	291			
$\hat{\rho}_c$:	.1168			

	120	119	
CO6 (43000061	.22	.41	
			CO6
Means:	.040	.044	.036
High:	.56, with 102		
Days:	224		
$\hat{\rho}_c$:	.1330		

	115	114	121	
IL2 (8520007)	.30	.38	.40	
				IL2
Means:	.041	.049	.049	.044
High:	.47, with 102			
Days:	402			
$\hat{\rho}_c$:	.0995			

TABLE 3

CORRELATIONS BETWEEN DAILY MEANS: NITRIC OXIDE

	119	111	110	
C01 (30001)	-.0034	-.024	-.0345	
119		.76	.68	
111			.71	
				C01
Means :	.012	.015	.007	.032
Highs: -0356, with 112				
Days: 200				
$\hat{\rho}_c$: .1407				

	113	120	121	
C02 (200002)	.027	-.011	.26	
113		.57	.52	
120			.35	
				C02
Means:	.0162	.0084	.0082	.023
High: .26, with 121				
Days: 116				
$\hat{\rho}_c$: .1841				

	112	120	
C04 (1040001)	.026	.097	
112		.62	
			C04
Means:	.021	.012	.038
High: .0972, with 120			
Days: 198			
$\hat{\rho}_c$: .1414			

	109	103	104	
IL6 (2120009)	-.007	.11	.094	
109		.53	.41	
103			.54	
				IL6
Means :	.0072	.011	.023	.036
Highs: .23, with 107				
Days: 343				
$\hat{\rho}_c$: .1077				

(Table 3, Continued)

	112	120	113	
co5 (4120001)	-.04	.06	-.06	
112		.57	.64	
120			.60	
				CI 1
Means:	.02	.01	.02	.024
High:	.06, with 120			
Days :	195			
$\hat{\rho}_c$:	.1425			

	102	107	113	
CI 2 (4280061)	.19	.21	.09	
102		.60	.62	
107			.70	
				CI 2
Means:	.024	.022	.014	.008
High:	.22, with 111			
Days:	206			
$\hat{\rho}_c$:	.1387			

	106	111	112	
CI 3 (4280062)	.12	.13.	.19	
106		.69	.59	
111			.73	
				CI 3
Means:	.019	.013	.016	.008
High:	.23, with 113			
Cays:	111			
$\hat{\rho}_c$:	.1881			

	101	105	106	
CI 5 (4280064)	.02	.028	-.026	
101		.45	.61	
105			.59	
				CI 5
Means:	.02	.026	.022	.024
High:	.19, with 115			
Days:	54			
$\hat{\rho}_c$:	.2673			

		120	119		CO6
CO6 (4300006)		.23	.14		
120			.62		
Means:		.011	.012		.023
High:	.32, with 103				
Days:	203				
$\hat{\rho}_c$:	.1397				

TABLE 5

CORRELATIONS BETWEEN DAILY MEANS: CARBON MONOXIDE

	119	111	110	
CO1 (30001)	.0036	.0256	-.077	
119		.59	.79	
111			.58	
				CO1
Means:	.74	.88	.60	.36
High:	.0545 with 120			
Days:	224			
$\hat{\rho}_c$:	.1325			

	113	120	121	
CO2 (200002)	-.065	-.12	-.023	
113		.55	.36	
120			.42	
				CO2
Means:	1.14	1.56	.26	.37
High:	.1053, with 104			
Days:	110			
$\hat{\rho}_c$:	.1890			

	112	120		
CO4 (1040001)	.15	-.12		
112		.15		
				CO4
Means:	.91	1.03		.34
High:	.15 with 112			
Days:	196			
$\hat{\rho}_c$:	.1421			

	104	103	105	
IL7 (2120008)	-.18	-.08	.075	
104		.13	.32	
103			.24	
				IL7
Means:	.80	2.04	5.16	.19
High:	.075, with 105			
Days:	16			
$\hat{\rho}_c$:	.4714			

(Table 5. Continued)

62

	109	103	104	
IL6 (2120009)	-.09	-.05	-.06	
109		.18	.33	
103			.19	
				IL6
Means:	.17	.68	.74	.15
High:	.076, with 108			
Days:	290			
$\hat{\rho}_c$:	.1170			

	112	120	113	
CO5 (412001)	.26	-.05	-.015	
112		.15	.12	
120			.55	
				C05
Means:	.86	1.06	1.05	.55
High:	.30, with 102			
Days :	156			
$\hat{\rho}_c$:	.1591			

	111	105	106	
CI1 (4280007)	-.07	-.06	-.10	
111		.38	.58	
105			.41	
				CI1
Means :	.71	.61	1.05	.22
High:	.09, with 104			
Days :	247			
$\hat{\rho}_c$:	.1267			

	102	107	113	
CI2 (4280061)	.13	-.05	.022	
102		.18	.28	
107			.26	
				CI2
Meals:	.44	1.83	.99	.29
High:	.135, with 102			
Days:	321			
$\hat{\rho}_c$:	.1113			

(Table 5, Continued)

Table 5, continued

	106	111	112	
CI 3 (4250062)	n. a.	n. a.	n. a.	
106		n. a.	n. a.	
111			n. a.	
				CI 3
Means:	1. 19	. 79	. 77	. 19
High:	n. a.			
Days:	141			
$\hat{\rho}_c$:	. 1672			

	113	114	101	
CI 4 (4280063)	. 08	. 04	. 13	
113		n. a.	n. a.	
114			n. a.	
				CI 4
Means:	1. 006	. 23	. 45	. 17
High:	. 18, with 105			
Days:	318			
$\hat{\rho}_c$:	. 1118			

	101	105	106	
CI 5 (4280064)	-. 23	-. 095	-. 26	
101		. 56	. 70	
105			. 41	
				CI 5
Means:	. 97	. 65	. 97	. 57
High:	. 03, with 112			
Days:	319			
$\hat{\rho}_c$:	. 1116			

	120	119	
C06 (4300006)	-. 23	-. 27	
120		. 88	
			C06
Means:	. 70	. 56	. 57
High:	. 03, with 112		
Days :	152		
$\hat{\rho}_c$:	. 1612		

(Table 5, Continued)

	115	114	121	
IL2 (8520007)	.0042	-.02	.019	
115		.46	.27	
114				.46
				IL2
Means:	.26	.24	.36	.11
High:	.1329, with 101			
Days:	289			
$\hat{\rho}_c$:	.1172			

TABLE 6

CORRELATIONS BETWEEN DAILY 90% DECILES: CARBON MONOXIDE

		119	111	110	
CO1 (30001)		.034	-.004	-.089	
					CO1
Means:		1.89	2.50	1.25	.486
High:	.034, with 119				
Days:	224				
$\hat{\rho}_c$:	.1330				

		113	120	121	
CO2 (200002)		-.06	-.12	.085	
					CO2
Means :		3.03	3.29	.90	.55
High :	.085, with 121				
Days:	110				
$\hat{\rho}_c$:	.1890				

		112	120		
CO4 (1040001)		.076	-.049		
					CO4
Means:		.277	2.40		.52
High:	.13, with 103				
Days:	197				
$\hat{\rho}_c$:	.1418				

		104	103	105	
IL7 (2120008)		-.14	.04	-.03	
					IL7
Means:		1.71	5.31	9.72	.26
High:	.13, with 107				
Days :	16				
$\hat{\rho}_c$:	.5055				

		109	103	104	
IL6 (2120009)		-.055	-.009	-.046	
					IL6
Means:		.69	1.46	1.99	.18
High:	.15, with 108				
Days:	318				
$\hat{\rho}_c$:	.1118				

	112	120	113	
C05 (4120001)	.07	-.047	-.042	
				C05
Means:	2.56	2.45	3.14	.87
High: .27, with 102				
Days: 156				
$\hat{\rho}_c$: .1591				

	111	105	106	
CI1 (4280007)	-.013	-.03	-.05	
				CI1
Means:	2.09	1.96	2.21	.41
High: .06, with 121				
Days: 248				
$\hat{\rho}_c$: .1265				

	102	107	113	
CI2 (4280061)	.15	-.014	.04	
				CI2
Means:	1.37	4.09	3.01	.51
High: .15, with 102				
Days: 323				
$\hat{\rho}_c$: .1109				

	106	111	112	
CI3 (4280062)	-.023	-.014	.02	
				CI3
Means:	2.17	2.16	2.52	.35
High: .066, with 102				
Days: 255				
$\hat{\rho}_c$: .1248				

	113	114	102	
CI4 (4280063)	-.001	-.005	.08	
				CI4
Means:	3.08	.81	1.39	.28
High: .098, with 121				
Days: 326				
$\hat{\rho}_c$: .1104				

Table 6, continued

	101	105	106	
CI5 (4280064)	-.11	-.006	-.11	
				CI5
Means:	2.57	1.99	2.20	.78
High:	.095, with 103			
Days:	319			
$\hat{\rho}_c$:	.1116			

	120	119		
CO6 (4300006)	.014	.019		
				CO6
Means:	1.82	1.64		.37
High:	.11, with 102			
Days:	154			
$\hat{\rho}_c$:	.1601			

	115	114	121	
IL2 (852000)	.008	-.006	-.07	
				IL2
Means:	.67	.83	1.04	.15
High:	.10, with 119			
Days:	317			
$\hat{\rho}_c$:	.1120			

TABLE 7

CORRELATIONS BETWEEN DAILY MEANS: SULFUR DIOXIDE

	119	111	110	
CO1 (30004)	n. a.	n. a.	n. a.	
119		n. a.	n. a.	
111			n. a.	
				CO1
Means:	n. a.	n. a.	n. a.	. 019
High:	. 09, with 114/. 05 with 106/. 07 with 101			
Days:	190			
$\hat{\rho}_c$:	. 1443			

	114	121	115	
IL1 (160006)	. 0017	. 078	-. 14	
111		. 16	n. a.	
121			. 10	
				IL1
Means:	. 012	. 008	. 010	. 029
High:	. 08, with 121			
Days:	53			
$\hat{\rho}_c$:	. 2697			

	113	120	121	
CO2 (200002)	-. 05	. 11	-. 006	
113		. 14	. 12	
120			. 09	
				CO2
Means:	. 013	. 018	. 015	. 014
High:	. 11, with 120			
Days:	120			
$\hat{\rho}_c$:	. 1181			

	112	120	
CO4 (1060001)	n. a.	. 018	
112		n. a.	
			CO4
Means:	n. a.	. 007	. 010
High:	. 21, with 113		
Days:	190		
$\hat{\rho}_c$:	. 1443		

Table 7, Continued)

	104	103	105	
IL7 (2120008)	.06	.024	.010	
104		.04	.0008	
103			.016	
				IL7
Means:	.032	.019	.020	.024
High:	.06, with 104			
Days:	396			
$\hat{\rho}_c$:	.1003			

	109	103	104	
IL6 (2120009)	n.a.	-.19	-.12	
109		n.a.	n.a.	
103			.04	
				IL6
Means:	n.a.	.009	.014	.025
High:	.176, with 114			
Days:	63			
$\hat{\rho}_c$:	.2481			

	108	103	102	
IL4 (29600012)	.12	-.14	n.a.	
108		.010	n.a.	
103			n.a.	
				IL4
Means:	.011	.009	n.a.	.024
High:	.13, with 114			
Days:	67			
$\hat{\rho}_c$:	.2408			

	112	120	113	
C05 (4120001)	n.a.	-.045	-.064	
112		n.a.	n.a.	
120			.20	
				C05
Means:	n.a.	.013	.019	.017
High:	.13, with 103			
Days:	194			
$\hat{\rho}_c$:	.1479			

(Table 7, Continued)

	102	107	113	
CI 2 (4280061)	n.a.	n.a.	.12	
102		n.a.	n.a.	
107			n.a.	
				CI 2
Means:	n.a.	n.a.	.009	.030
High:	.32, with 114			
Days:	41			
$\hat{\rho}_c$:	.3050			

	113	114	102	
CI 4 (4280063)	-.027	-.077	n.a.	
113		.11	n.a.	
116			n.a.	
				CI 4
Means:	.0085	.014	n.a.	.024
High:	.043, with 121			
Days:	43			
$\hat{\rho}_c$:	.2981			

	115	114	121	
IL2 (8520007)	-.048	-.105	.035	
115		.26	.10	
114			.16	
				IL2
Means:	.0096	.012	.0073	.042
High:	.17, with 106			
Days:	65			
$\hat{\rho}_c$:	.2443			

TABLE 8

CORRELATIONS BETWEEN 90% DECILES: SULFUR DIOXIDE

	119	111	110	
C01 (30001)	n. a.	n. a.	n. a.	
				C01
Means:	n. a.	n. a.	n. a.	.40
High:	.14, with 105/.06, with 108/.087 with 114			
Days:	190			
$\hat{\rho}_c$:	.1443			

	114	121	115	
IL1 (160006)	-.027	-.06	-.12	
				IL1
Means:	.04	.021	.036	.072
High:	.025, with 108			
Days:	53			
$\hat{\rho}_c$:	.2697			

	113	120	121	
C02 (200002)	-.09	.033	-.075	
				C02
Means:	.043	.037	.055	.030
High:	.033, with 120			
Days:	120			
$\hat{\rho}_c$:	.1811			

	112	120	
C04 (1040001)	n. a.	.059	
			C04
Means:		.025	.025
High:	.17, with 113		
Days:	190		
$\hat{\rho}_c$:	.1443		

	104	103	105	
IL7 (2120008)	-.003	-.002	.06	
				IL7
Means:	.086	.045	.049	.062
High:	.066, with 114			
Days:	396			
$\hat{\rho}_c$:	.1003			

(Table 8, Continued)

	109	103	104	
IL6 (2120009)	n. a.	-. 22	-. 068	
				I16
Means:	n. a.	. 025	. 035	. 057
High:	. 27, with 120			
Days:	63			
$\hat{\rho}_c$:	. 2481			

	108	103	102	
IL4 (2960012)	. 21	-. 18	n. a.	
				IL4
Means:	. 036	. 025	n. a.	. 051
High:	. 21, with 108			
Days:	67			
$\hat{\rho}_c$:	. 2408			

	112	120	113	
C05 (4120001)	n. a.	. 041	. 04	
				C05
Means:	n. a.	. 032	. 063	. 035
High:	. 19, with 103			
Days:	194			
$\hat{\rho}_c$:	. 1429			

	102	107	113	
CI2 (4280061)	n. a.	n. a.	-. 01	
				CI2
Means:	n. a.	n. a.	. 028	. 069
High:	. 57, with 114			
Days:	41			
$\hat{\rho}_c$:	. 3050			

(Table 8, Continued)

Table 8, continued

		113	114	102	
CI4		-.08	-.07	n.a.	
(4280063)					CI4
Means:		.028	.045	n.a.	.061
High:	-.0015, with 104				
Days:	43				
$\hat{\rho}_c$:	.2981				

		115	114	121	
IL2		.041	-.13	.086	
(8520007)					IL2
Means:		.032	.038	.017	.10
High:	.21, with 106				
Days:	65				
$\hat{\rho}_c$:	.2443				

FOOTNOTES

1. In particular, discussions with August Auer, Professor of Atmospheric Science, University of Wyoming, Laramie.

CHAPTER 6

EMPIRICAL ESTIMATION OF THE MODEL

1. Introduction

     This chapter presents estimates of the willingness to pay for
reductions in ozone levels in St. Louis. The methodology used in making
these calculations focuses on estimating the marginal bid expression
derived in Chapter 3

$$dB/d\alpha = H_\alpha q_M / H_M \qquad (1)$$

where H_α denotes the marginal effect of a change in air pollution on the
stock of health, H_M denotes the marginal effect of a change in the
consumption of medical services on the health stock, and q_M denotes the
full price of medical care. Section 2 discusses two alternative approaches
that could be used to estimate equation (1) and selects one of these for
empirical implementation. Section 3 shows how the variables used in the
empirical analysis were constructed from the information given in the St.
Louis health survey and the RAMS data. (The St. Louis health survey also
is described in detail in Appendix 3.) Section 4, then, presents the
results. More specifically, this section gives estimates of the health
production function as well as calculations of the marginal willingness to
pay for reduced ozone levels. Section 5 offers a brief summary.

2. Estimation of the Willingness to Pay

     As indicated, this section surveys two possible approaches to
estimating equation (1). The first approach seeks to obtain separate
estimates of H_α and H_M from the health production function based on
a procedure similar to that used by Rosenzweig and Schultz (1982a, 1982b).
Rosenzweig and Schultz, who examined the health of children rather than
adults, used birthweight as an indicator of the heaith stock.
Unfortunately, from the St. Louis health data set, no single comprehensive
parallel measure of the size of an adult's health stock can be constructed.
Information concerning subjectively reported health status (excellent,
good, fair, poor), existence of specific types of chronic conditions, and
length of suffering from chronic conditions is available for each
individual; however, these variables alone may not accurately measure the
theoretical concept of the stock of health. This point will be discussed
more fully momentarily. Nevertheless, the conclusion drawn is that the
stock of health may be best treated as a multi-dimensional, rather than a
single dimensional variable. That perspective underlies the second
approach to obtaining $dB/d\alpha$, in which only the ration H_α / H_M is estimated.

A.   The Rosenzweig and Schultz Approach

        The Rosenzweig and Schultz method for estimating the health
production uses a two stage procedure. First, a reduced form demand
equation for the health input, M,is derived from the model as shown in
equation (2) and empirically estimated

$$M = M(q_X, q_M, W, \alpha) \qquad\qquad (2)$$

All variables in those equations are defined as in Chapter 3. The fitted
values of M then are used in the second stage in order to estimate the
health production function

$$H = H(M; \alpha) \qquad\qquad (3)$$

The idea behind using this procedure is to explicitly account for the
interaction between the choice variables H and M and to obtain
statistically consistent estimates of the health production function. In
other words, the health of an individual is determined by the level of the
health inputs used in the production of health and other exogenous factors.
The levels of the health inputs are, in turn, determined by the individual
on the basis of all relevant information known to him. Because of the
interaction of the demand for health inputs and the production of health,
simultaneity exists in this system of equations, and thus the whole system
of equations must be estimated in order that the resulting coefficients are
consistent. A two stage procedure which utilizes the estimated input
demand equations to calculate fitted values for the health inputs and then
uses these fitted values in place of the actual values when estimating the
health production function would eliminate this inconsistency.

        In order to estimate a production function for health in this manner,
a variable that measures the stock of heaith must be available. This
presents a problem since the stock of health is a theoretical concept and
not easily quantified. Rosenzweig and Schultz used birthweight as a proxy
for the health stock of children; however, as previously indicated, the
available health stock measures pertaining to adults in the St. Louis data
probably are not as good. In any case, the following subsection provides a
critical evaluation of the health information that the St. Louis data set
contains.

B.   The St. Louis Health Data

        Three measures are available from the St. Louis data set that
directly pertain to an individual's stock of health. These are the
categorical self-evaluation of health; i.e., reported health status is
excellent, good, fair, or poor; the number and types of chronic conditions
or illnesses; and the length of time the individual has suffered from these
conditions on illnesses. Each of these measures have been used in other
empirical health related analyses; although none of them are without
limitations For example, the self-evaluation of health is a highly
subjective measure. What is considered excellent health to one individual
may only be good health to another. Moreover, someone with a chronic

illness may consider himself to be in good health as does someone who gets several colds a year. But do both of these individuals actually have the same health stock or is there an important difference in their health status? Another problem with this measure is how to quantify the four possible responses. How much more health stock does an individual in excellent health possess as opposed to an individual in poor health? This measure could be expressed as a categorical variable but much information on the actual health stock would be lost.

Additionally, expressing the health stock of an individual as a function of the number and types of existing chronic illnesses also presents significant problems. One problem was mentioned in the previous paragraph. How can comparisons be made between the health stock of an individual with a chronic illness and the health stock of an individual with no chronic illness? Is it true that the presence of a chronic illness implies that the individual possesses a smaller health stock than those without any chronic illnesses? Compare, for example, the person who is rundown, gets frequent colds, and feels tired much of the time to the person who has asthma but takes very good care of his health.   In this case, the absence of a chronic illness does not necessarily imply a larger health stock.

Finally, the duration of chronic illnesses is most meaningful when used in conjunction with information on the number and types of such health problems. Without knowing the condition from which the individual is suffering, information on the length of time it has been present probably tells little about the magnitude of the health stock.

A further problem with each of these variables taken separately is that they each may be measuring different dimensions of the health stock. To illustrate, if the available three variables were comprehensive measures of the health stock, then they should be highly correlated. That situation, however, does not materialize in the St. Louis data set as shown in Table 1. The Pearson correlations between possible health stock variables does have a plausible sign pattern in that the incidence of chronic conditions is negatively associated with the excellent health variables and positively associated with the poor health variables. Nevertheless, the linear associations between these variables are not particularly strong. CHRO and POOR have a Pearson correlation of .313 and CHRO and EXCELLENT have a corresponding correlation of -.295. The correlations between the subjective health evaluation variables and the duration of chronic illness measures are quite similar to those shown in Table 1 since that latter variable is either zero or non-zero whenever the incidence of chronic illness variable is zero or non-zero.

On the basis of this discussion, assigning great faith in the available measures of the health stock probably is unwarranted. Nevertheless, these measures easily may provide greater information about the health stock when used together, rather than when used separately. In other words, if a choice must be made as to which single variable is best suited to serve as a proxy for the health stock in a regression equation, there would be little on which to base it. As a

TABLE 1

PEARSON CORRELATIONS BETWEEN POSSIBLE HEALTH STOCK VARIABLES

	CHRO	RESPCIRC	OTHERCH
POOR	.3125	.1633	.256
FAIR	.2894	.2401	.1804
GOOD	.0380	.0561	-.0001
EXCELLENT	-.295	-.213	-. 197

*
 Variable definitions are:

    CHRO: Indicates presence of a chronic illness
    RESPCIRC: Indicates presence of a respiratory or circulatory
                   illness
    OTHERCH: Indicates presence of a chronic illness other than of a
                   respiratory or circulatory nature
    POOR: Indicates respondent is in poor health
    FAIR: Indicates respondent is in fair health
    GOOD: Indicates respondent is in good health
    EXCELLENT: Indicates respondents is in excellent health

consequence, the design of the estimation strategy presented below shows
how multiple indications of the health stock can usefully be incorporated
into the equation to be estimated.

C.   An Alternative Approach

A suitable alternative approach to that proposed by Rosenzweig
and Schultz for estimating the quantity dB/da easily can be obtained under
the assumption that the health production function is approximately linear.
The assumption of linearity in logarithms or some other simple function
also could be applied with out affecting the nature of the approach
considered. Let

$$H_i = \beta_0 + \beta_1 M_i + \beta_2 \alpha_i + U_i \tag{4}$$

where the subscript i denotes the ith individual and U_i denotes a random
disturbance capturing unmeasured variables affecting H_i. To more easily
allow for the fact that H_i may be best measured by a set of health
indications rather than a single variable rewrite equation (4) as

$$M_i = -(\beta_0/\beta_1) + (1/\beta_1)H_i - (\beta_2/\beta_1)\alpha_i + V_i \tag{5}$$

where the new disturbance V_i can be expressed as

$$V_i = -U_i/\beta_1 \qquad\qquad (6)$$

Two features of equations (4), (5), and (6) are worth elaborating. First, in equation (5), the coefficient of the air quality variable α_i is just the negative of the analogue to H_α/H_M from the theoretical model? Therefore, when equation (5) is **empirically** estimated, the negative of the coefficient on air quality need only be multiplied by q_M, the full price of medical care, in order to find $dB/d\alpha$. Second, (4) is likely to be an overidentified equation from the structural system to which it belongs. The other three structural equations for the theoretical model determining M_i, X_i, and the value of the Lagrange multiplier, λ_i, would contain the predetermined variables, q_{Xi}, q_{Mi}, and W_i. Those prices would not enter the production function. **Moreover, equation** (4) contains an air quality measure which would not appear in the other three structural equations. As the theoretical model demonstrates, air quality affects the values of the choice variables only through its impact on health.

The remainder of this chapter is concerned with estimating equation (5) and the expression for $dB/d\alpha$ using the St. Louis health and the RAMS air quality data. Section 3 shows how the sample on which these estimates are based was constructed and how the variables in (5) were defined. Section 4 presents the empirical results.

3. Sample Construction and Variable Definitions

A. Sample Construction

For the purpose of this study only these individuals whose major activity was recorded as employed were included in this sample. (See Appendix 3 for a more complete description of these data.) The reason for excluding all others was that no data were available to assess their value of time; a necessary ingredient in computing the full prices. The value of time to an employed individual is imputed to be the wage rate and no comparable measure is available for others who may be retired, too young to work, etc. In order to calculate this measure, more detailed information is required. Of the 2197 employed persons in the survey, only 820 provided hourly wage data.

B. Variable Definitions

The variables used in the empirical analysis can be divided into five categories, those measuring: (1) consumption of medical services, (2) the price of medical services, (3) the wage rate, (4) socioeconomic-demographic characteristics, and (5) air quality. Each of these categories is considered sequentially.

1) Medical Care

Existing information in the St. Louis health data set measuring the consumption of medical services certainly are far from perfect. What is desired here is a variable that would measure whether an individual

received regular medical check ups; however, there was no question in the survey that explicitly answered this question. The number of times the individual visited a doctor during the year preceding the Individual Background Interview (1977 or 1978 depending on when the respondent was enrolled in the survey) and a yes/no question asking if a doctor was usually seen at least once a year were recorded for each individual. If an individual had a rash of illnesses or developed a chronic illness the year preceding survey, his answer to the first question would be biased upward for this would not have been his response in a "typical" year. His response would be measuring the amount of time spent ill rather than whether he sought preventive medical care. Or as another example, a pregnant woman may visit the doctor every two weeks toward the end of her term; therefore, her response will be much higher than it would have been the preceding year and it may in fact measure the health of her child.

The second question still does not measure preventive medical care; however it may come closer than the first question. That "usually" is included in the question at least partially controls for any bias that may come from an individual experiencing an atypical year for doctor visits. Those who have visited the doctor for annual check ups should respond affirmatively to this question in addition to those who visited a physician for the treatment of disease only. Even if an individual has seen the doctor for a specific treatment he will have received more preventive medical care than a person who has received no medical care at all for a physician may notice other physical problems in the process of treatment or he may give the patient advice on preventive measures necessary to stay healthy. Thus, the second question may measure preventive medical care more accurately than the first.

2) Price of Medical Care

The price of medical care was constructed to take into account both direct dollar outlays for the medical care, the time costs involved in commuting to and from the source of medical care, and the waiting time at the source of medical care. The only available information on the direct dollar outlays was a question regarding the usual charge by the doctor for an office visit. Many individuals have health insurance which pays for part or all of their office visits, thus their actual dollar outlay may be much lower than that which is recorded for the office charge. The only data available which may alleviate this problem are obtained from a question asking the portion of an office visit paid by any health insurance. Unfortunately, the answers to this question are categorized very generally (all or most, some or about half, little or none) and provide little useful information. With these difficulties in mind, the price of medical care is defined as:

q_M = Office Visit Charge + hourly wage (commuting time

+ office waiting time)   (7)

where commuting time + office waiting time are measured in hours. The data on usual office visit charges and hourly wage rates came from the

supplemental survey and therefore are in 1980 dollars. Note that q_M probably overestimates the true cost per visit which individuals face since no account is taken of any insurance reimbursement of medical fees. Moreover, the hourly wage rate also may overstate the value of time.

3) Wage Rate

As shown in equation (7), the hourly wage rate is used as the value of time in computing the price of medical care. That wage rate is defined as follows. For the entire eight week follow-up period (in 1978 or 1979), individuals kept track of where they were during each 24 hour day. The data thus gives a record of the number of hours worked during the entire eight weeks. From this record an average number of hours per week was computed. Also, in the supplemental survey (1980) was a question regarding take home pay from an individual's full time occupation and the pay period corresponding to this amount. The amount of take home pay per week are computed from these two items and by dividing this figure by the average number of hours worked per week a variable measuring the hourly wage was constructed. As indicated in the previous subsection, data on take home pay was missing for a substantial fraction of workers.

4) Socioeconomic-Demographic Variables

In this chapter, the α variable has frequently been discussed, for the sake of simplicity, as if it referred exclusively to changes in air quality. However, in the model presented in Chapter 3, a was defined to be a vector of exogenous variables that affect the efficiency with which medical services are used in producing the health stock. This subsection makes that specification more explicit by stating which additional variables also might be included. These are: (1) age, (2) education, (3) race, and (4) sex.

Age, simply given by years of age, is included to reflect the loss of efficiency in producing the health stock as the individual grows older. Education is the number of years of schooling completed by the individual. Education is used here to measure human capital, which may affect how efficiently that person will combine inputs to produce a given stock of health. There is some difficulty when education is used to measure human capital, however. The stock of human capital will not be constant over a person's lifetime as suggested by the use of education. Instead, it will tend to increase as an individual receives on-the-job-training and more informal education and it can also depreciate as a person grows older. The use of years of schooling also poses some difficulties as the number of years of schooling does not always correspond directly with years of education. Thus, there are at least two sources of measurement error when years of schooling is used to measure education but there is probably an equal chance that the error is negative as it is positive. Finally, race and sex are both used as exogenous variables to test the hypothesis that one race or one sex may be more efficient producers of health than the other therefore their demand for the health inputs may vary accordingly.

5) Air Quality Variables

The only remaining exogenous variables are those measuring air quality in the St. Louis area. As discussed earlier this posed some difficulty because of the inopportune timing of the survey and the collection of the RAMS air pollution data. Had the RAMS air pollution data and the survey been conducted at the same time, it would have been possible to measure the relationship between work loss days and the amount of air pollution in the area on those same work loss days or the days just prior to them. The hypothesis being tested in this case would be whether people adjust their behavior on a day to day basis. In order to test this, it would also be necessary to have daily information on an individual's personal habits besides the daily air pollution readings and the record of work loss days and when they occurred.

If it is assumed that the yearly averages of each pollutant for each station are "typical" for any given year these yearly averages can be used as a measure of air quality for the time that the survey took place. The use of yearly data for one particular area poses a difficulty for this analysis. This study proposes to test the hypothesis that individuals may compensate for a decrease in air quality by adjusting their health related behavior. By using only one SMSA area and using annual data the only variation in air quality will arise from certain areas of the SMSA having higher concentration of air pollution than others. This variation may still be quite large. For example, areas that are congested with rush hour traffic normally have a much higher concentration of photochemical oxidants than areas that experience little peak hour traffic. But if annual data are used, it would be better to have observations on more areas in order to obtain a broader range of pollution exposures.

The pollution variables include the mean levels of ozone, sulfur dioxide, oxides of nitrogen, small (inhalable) total suspended particulates, and small (inhalable) suspended lead particulates. Other pollution variables were available but these represent most aggregate measures of pollutants. In Appendix 1, synergisms between ozone and other pollutants were discussed. Researchers have found evidence that ozone in combination with sulfur dioxides and nitrogen oxides may be more harmful to human health than any of these pollutants taken alone. Therefore, it is necessary to include these air quality measures in the list of independent variables. These variables are constructed by simply multiplying the two relevant pollutants together. Also constructed was a variable combining-mean levels of ozone and TSP in order to account for any synergistic effects between these two pollutants.

4. Empirical Results

In this section, estimates of the willingness to pay for improved air quality are presented using the methodology underlying equation (5) for two separate samples of St. Louis workers. The first sample is composed of the 820 persons for whom wage information is available and the second sample is composed of 2197 workers. For both of these samples, the equation to be

estimated is shown in equation (8)

$$MED_i = MED(\alpha_i, AGE_i, EDUC_i, SEX_i, RACE_i, H_i) \qquad (8)$$

where α_i now denotes a set of air quality measures and H_i denotes a set of measures of the health stock. Additionally, each of the variables used to estimate equation (8) is defined in Table 2. That table also gives the arithmetic mean for each variable in each of the two samples. Note that the categorical self-evaluation of health variable is not listed in Table 2. Preliminary estimates of equation (8) using that variable produced poor results and consequently it simply was dropped from further consideration.

Moreover, given the variable definitions in Table 2, the components of α_i are measured as levels of air pollution rather than as measures of air quality. Hence, the expected signs on the coefficients of α_i would be positive and those coefficients can be used directly, without multiplying by minus one, in computing $dB/d\alpha$. Given the manipulation used to derive equation (8), the expected signs on CHRO and Length should be negative since increases in these variables are associated with decreases in the health stock. Also, the expected signs on the four socioeconomic-demographic variables are as follows: (1) the coefficient of AGE would be positive if the aging process reduces the efficiency with which the health stock is produced, (2) the coefficient of EDUC would be negative if years of schooling increase the efficiency with which health is produced and (3) the coefficients of SEX and RACE should be positive if males and blacks tend to have lower health stocks. Several measures of air quality were tried as independent variables. Since ozone was of interest it was included in all equation specifications. Measures of lead content in the air were also included in some of the specifications to test the findings reported in Appendix 2. Sulfur dioxides and total suspended particulates have also been found to cause deleterious health effects; therefore they were included in some specifications. In Appendix 1, it was mentioned that synergisms among pollutants may be more harmful than each pollutant taken separately. Several researchers found evidence that ozone and sulfur dioxides and ozone and nitrogen oxides could combine synergistically to produce more harmful effects to human health than does ozone alone. Therefore these two synergisms were included in several of the specifications.

The decision of which pollutants to include depended partially on the correlations among pollutants. The only correlation found to be particularly high was SULDIOM with OXSULM. Care was taken not to include two highly correlated air quality measures in the same equation so as to make the results more reliable. Pearson correlations between the pollution and nonpollution variables as well as between the nonpollution variables themselves were on the order of .20 or smaller and certainly not high enough to suggest a serious multicollinearity problem.

Because of the discrete nature of the dependent variable, MED_i, and the inclusion of the health stock (a choice variable) as a covariate, a simultaneous equation logit model was developed for each equation estimated. Predicted values for CHRO and LENGTH were obtained from their

84

TABLE 2

VARIABLE DEFINITIONS*

VARIABLE	DEFINITION	SAMPLE SIZE 820 MEAN	SAMPLE SIZE 2197 MEAN
MED	1: Denotes respondent sees a doctor at least once annually	.747	.755
PMED	Price of medical care	40.744	45.38
HWAGE	Hourly wage	5.078	5.078
RACE	1: Indicates person is black	.277	.241
AGE	Age in years	39.28	40.33
EDUC	Years of schooling completed	12.65	12.79
SEX	1: Indicates respondents is male	.539	.583
OZONEM	Mean ozone level (ppm)	.019	.019
SULDIOM	Mean sulfur dioxide level (ppm)	.024	.024
TSPSMLM	Total suspended particulates, small size, mean levels, (micrograms/m^3)	22.81	23.77
LEADSMLM	Lead, small size, mean (micrograms/m^3)	706.084	705.42
OZSULM	OZONEM x SULDIOM	<.000	<.000
OZNITM	OZONEM x OXNITM	<.000	<.001
CHRO	1: Denotes respondent reports presence of a chronic illness	.105	.108
LENGTH	Number of years respondent has had chronic illness	1.374	1.327

*
See Appendix 3 for more complete details.

respective reduced form equations and then substituted into equation (8).
Since CHRO also is a discrete variable, its reduced form equation, which
uses $PMED_i$, $HWAGE_i$, α_i, SEX_i, $RACE_i$, AGE_i, and $EDUC_i$ as covariates, is
estimated in a logit framework. The reduced form equation for LENGTH, on
the other hand, which uses the same covariates, is estimated as a Tobit
model. The frequency distribution of the length variable is characterized
by a large number of zeros (more than half of the values are zero) and then
integer values ranging as high as 27.

From an econometric viewpoint, the simultaneous equation system
described above certainly is not a typical one in view of the fact that the
dependent variables either are discrete or truncated. Nelson and Olson
(1978), however, have demonstrated that the procedure used here, which is
analogous to two-stage least squares, produces consistent and
asymptotically normal estimates. Furthermore, on the basis of a small
sample simulation experiment, those same authors conclude that estimated
standard errors for the reported coefficients tend to be biased upward.
Therefore, tests for whether those coefficients are statistically
significant would be conservative.

When estimating equation (8) in the simultaneous equation logit
framework, the probability, P_j, that the jth individual falls into the
category that is assigned a value of one is equal to:

$$P_j = \exp\{Z_j\beta\}/[1 + \exp\{Z_j\beta\}] \tag{9}$$

where Z_j is the jth individual's vector of covariates and β is a
coefficient vector. In this model, the marginal effect of the kth
independent variable on P_j is given by:

$$\frac{\partial P_j}{\partial Z_{jk}} \quad \beta_K P_j(1 - P_j) \tag{10}$$

Thus, by estimating equation (8) in the form shown in (19), the effect of a
change in air pollution levels on the probability of usually seeing a
doctor at least once per year can be calculated. That effect corresponds
to the H_α/H_M term in the willingness to pay expression, $dB/d\alpha$. However,
the correspondence is rather inexact since MED_i is a dummy variable,
whereas PI in the theoretical model measures the quantity of medical
services consumed. Also, since MED_i indicates whether a doctor is usually
seen at least once per year, the resulting estimate of H_α/H_M probably
understates the true value of that parameter; a factor that would offset
the overestimate of q_M discussed previously.

The marginal effects evaluated at the mean of each independent
variable are reported in Tables 3 and 4. In parentheses are the χ^2
statistics (distributed with one degree of freedom) for each variable.
These statistics test the null hypothesis that the marginal effect of the
variable in question is equal to zero. Recorded below each column of
derivatives are: (1) the number of cases (NC), (2) the χ^2 statistic (with
degrees of freedom) for the equation as a whole which is useful in testing

TABLE 3

ESTIMATES OF THE HEALTH PRODUCTION FUNCTION
(DERIVATIVES EVALUATED AT THE MEAN)
820 CASES

	1	2	3	4	5	6
ONE	−.064	−.307	−.040	−.010	−.290	.226
	(.046)	(.269)	(.018)	(.001)	(.231)	(.392)
OZONEM	30.52**	33.14**	31.25**	29.74**	33.06**	25.72*
	(5.04)	(5.15)	(5.40)	(4.86)	(4.86)	(3.31)
SULDIOM		−.416	.390			
		(.055)	(.042)			
TSPSMLM		−.009			.008	
		(.322)			(.266)	
LEADSMLM						−.0003
						(2.07)
OXSULM					-8.82	95.94
					(.009)	(.785)
OZNITM			-91.90	-78.12		
			(.424)	(.370)		
AGE	−.003	−.003	−.004	−.003	−.003	−.003
	(1.34)	(1.66)	(1.96)	(1.56)	(1.61)	(1.16)
EDUC	−.012	−.011	−.009	−.010	−.012	−.013
	(1.29)	(1.37)	(.949)	(.975)	(1.50)	(1.84)
SEX	-.198***	-.200***	-.202***	-.199***	-.1999***	-.196***
	(29.62)	(30.46)	(30.73)	(29.84)	(30.29)	(29.14)
RACE	.064	.065	.079*	.077*	.065	.069
	(2.12)	(2.19)	(3.01)	(3.00)	(2.60)	(2.44)
$\bar{\text{CHRO}}$	−.100	−.036	.203	.044	−.078	−.248
	(.008)	(.001)	(.036)	(.002)	(.006)	(.055)
$\hat{\text{LENGTH}}$	.049	-050	.041	.042	.052	.057
	(1.11)	(1.20)	(.823)	(.808)	(1.32)	(1.68)
NC	820	820	820	820	820	820
χ^2 (df)	51.1(7)***	51.7(9)***	51.7(9)***	51.3(8)***	51.7(8)***	53.3(9)***
FCP	618	618	618	618	618	618

***denotes significance at 1% level.
 **denotes significance at 5% level.
  *denotes significance at 10% level.

TABLE 4

ESTIMATES OF THE HEALTH PRODUCTION FUNCTION
(DERIVATIVES EVALUATED AT THE MEAN)
2197 CASES

	1	2	3	4	5	6
ONE	-.793***	-1.38***	-.936***	-.949***	-1.32***	-1.08***
	(26.58)	(23.73)	(20.92)	(24.18)	(22.40)	(23.24)
OZONEM	53.85***	56.64***	54.28***	55.86***	53.64***	55.13***
	(52.53)	(52.86)	(44.66)	(49.02)	(52.46)	(50.58)
SULDIOM		2.97**	2.73**			
		(6.04)	(5.03)			
TSPSMLM		.020**			.020**	
		(6.60)			(6.59)	
LEADSMLM						.0003**
						(5.48)
OXSULM					151.71**	91.04
					(6.04)	(1.79)
OZNITM			121.65	197.94***		
			(2.10)	(7.14)		
AGE	-.012***	-.011***	-.011***	-.011***	-.011***	-.011***
	(41.51)	(39.85)	(35.41)	(39.76)	(40.4)	(39.84)
EDUC	.005	.005	.004	.004	.005	.004
	(1.26)	(1.44)	(.957)	(.975)	(1.44)	(.899)
SEX	-.274***	-.307***	-.300***	-.313***	-.311***	-.306***
	(119.94)	(119.40)	(113.29)	(117.2)	(119.6)	(118.9)
RACE	.101***	.105***	.094***	.088***	.106***	.104***
$\hat{~}$	(15.51)	(16.16)	(12.89)	(11.82)	(16.52)	(15.87)
CHRO	5.71***	5.19***	4.85***	4.88***	5.20***	5.12***
	(41.08)	(42.46)	(36.84)	(40.69)	(42.96)	(41.98)
$\hat{~}$ LENGTH	-.035	-.033	-.027	-.030	-.033	-.028
	(1.52)	(1.49)	(.979)	(1.16)	(1.51)	(1.16)
NC	2197	2197	2197	2197	2197	2197
χ^2(df)	149.5(7)	152.8(9)	146.4(9)	149.59(8)	153.1(9)	151.1(9)	114
FCP	1669	1676	1668	1667	1678	1668

***denotes significance at 1% level.
 **denotes significance at 5% level.
  *denotes significance at 10% level.

the null hypothesis of no relationship between the dependent and
independent variables, and (3) the fraction of cases correctly predicted by
the equation (FCP).

Table 3, which provides the estimates of the health production
function based on the 820 cases for which wage data are available, shows
that $\hat{CHRO}$ has the correct sign but is not significantly different from
zero at conventional levels. $\hat{LENGTH}$ is neither correctly signed nor
significantly different from zero. One explanation for this outcome for
these variables may lie in the poor fits achieved by the reduced form
equations for CHRO and LENGTH. With respect to the performance of the air
pollution variables, however, the derivative of OZONEM is positive and
significant at either the 5 percent level or the 10 percent level in all
six equations. Additionally, the value of the derivative of OZONEM range
from 25.72 to 33.14 indicating that the effects are not particularly
sensitive to the choices made concerning which other pollution variables to
include. Qualitatively at least, the results tend to corroborate the
evidence presented in Appendix 1. That material reported some of the
physiological effects of ozone exposure to be impaired pulmonary function
and sensory irritation such as burning eyes or scratchy throat; i.e.,
exactly the type of health effects for which medical attention might be
sought. The other pollution variables, however, did not perform nearly as
well. In all six equations, no air quality derivative except the one for
OZONEM was significantly different from zero at even the 10 percent level.
In fact, the derivative of LEADSMLM was the wrong sign and insignificant.
In the present context, however, that result is plausible since the health
effects of lead poisoning, as indicated in Appendix 2, are of a long term
nature and do not necessarily result in symptoms requiring immediate
medical attention. Low-level lead poisoning, for example, may lead to
progressive disorders of the cardiovascular, renal, or neurological
systems, or simply to a general dullness or irritability. Finally, none
of the possible synergistic effects examined had significant
derivatives.

Among the socieoeconomic-demographic variables, AGE entered
consistently with the wrong sign and its derivative never was significant.
That result may seem somewhat surprising, however, in a sample composed
only of employed workers, there may not be sufficient variation in years of
age to capture effect of this variable on the health stock. Moreover, the
relation between age and the health stock may be strongly non-linear, with
the largest effects occurring among those at the highest ages; i.e., those
not well represented in the sample considered here. EDUC, on the other
hand, was always the correct sign (negative) implying those with more
schooling are more efficient producers of health. The derivative of EDUC,
however, never was significantly different from zero. The derivatives of
SEX and RACE indicate that in the sample considered females and blacks tend
to have lower health stocks than males and whites respectively. Both
derivatives are consistently and significantly different from zero, with
the derivative of SEX always being significant at the 1 percent level.

89

Table 4 provides parallel results for the 2197 observation sample, which as has been indicated repeatedly, suffered from a severe incidence of missing data on hourly wages. The sign pattern of the derivatives of CHRO and LENGTH reversed in the larger sample-as compared with the situation found in Table 3. Thus, the sign for CHRO reported in Table 4 is wrong and the sign for LENGTH is correct. Distressingly, the derivative of CHRO is highly significant. The air pollution variables, on the other hand, performed better in the equations shown in Table 4 than in those shown in Table 3. The derivative of OZONEM is always significant at the 1 percent level. In addition, the derivatives of the remaining five pollution variables always are of the correct sign and generally are significant at the 5 percent level. That statement applies in particular to the coefficient of LEADSMLM shown in column 6. Thus, these results suggest that the health stock responds negatively to increases in concentrations of air pollutants in addition to ozone and that the synergism discussed in Appendix 1 may be important to consider (see columns 4 and 5). The socieoeconomic-demographic variables performed similarly with the results reported in Table 3, although the significance levels for the derivatives of AGE, SEX, and RACE were considerably higher.

With some trepidation, the results from Tables 3 and 4 can be used to make some illustrative willingness to pay estimates for a reduction in ozone levels. These benefit estimates are offered advisedly largely because of two problems, discussed previously, in calculating $dB/d\alpha$: (1) the dependent variable in the equations estimated is only an indicator of whether medical care was received and does not measure the level of consumption of medical services and (2) the price of medical care employed in the calculations may tend to overstate the price actually faced by the individuals in the sample. In any case, since ozone was the only air quality variable that performed consistently well, in the 820 observation data set and since it is of primary interest in this study, only reductions in that pollutant are used in making the benefit calculations. Because St. Louis experiences only a comparatively small number of days a year when the hourly average exceeds the national primary and secondary standards it will not take a large reduction in ozone levels to meet the standard. Therefore, reductions in the ozone level of 10 percent, 15 percent, 20 percent, and 30 percent of the mean ozone level (.019) have been used to calculate the benefits.

Willingness to pay estimates are presented based on the two equations presented in columns 3 in both Tables 3 and 4. Those equations had neither the highest and lowest OZONEM derivatives; therefore, the estimates presented would typify the results obtained from the remaining equations. Since the MED variable reflects whether a doctor usually is seen at least once per year, the willingness to pay estimates also would be annual figures. Moreover, all of the willingness to pay estimates reported are computed for individual "average" employed worker. In other words, the means of all independent variables, provided in Table 2, were used in the empirical approximation to H_α/H_M, as was the mean the price of medical care variable, q_M. Table 5 presents the willingness to pay calculations.

TABLE 5

WILLINGNESS TO PAY FOR REDUCTIONS IN OZONE LEVELS

| | PERCENT REDUCTION IN MEAN OZONE LEVELS | | | |
EQUATION	10%	15%	20%	30%
3 (Table 3)	$2.42	$3.63	$4.84	$7.27
3 (Table 4)	$2.69	$4.04	$5.39	$8.08

The estimates of willingness to pay for a 10 percent (.0019 ppm) reduction in mean ozone levels range from $2.42 to $2.69. For a 30 percent (.0057 ppm) reduction in mean ozone levels the willingness to pay estimates range from $7.27 to $8.08. It should again be stressed that these estimates are in annual terms and pertain to the "average" worker in St. Louis. Although these willingness to pay figures may appear to be small, they still are larger than those found by Seskin in his study of photochemical oxidant levels and acute illness in the Washington, D.C. area. Chapter 2 reported Seskin's finding that a 55.6 percent reduction in maximum one-hour average 1973 oxidant levels, necessary to meet the 1971-78 national standard, would result in a savings to Group Health Association plan members of $4490 in that year. Since during 1973, there were roughly 100,000 GHA members, annual benefits per member would have been, approximately $.04. Seskin's benefit estimate rises somewhat for 1974. During that year a 42.9 percent reduction in photochemical oxidant levels would have been necessary to meet the national standard; a reduction that would have resulted in benefit for GHA members of $12,140 or about $.12 per member. As noted in Chapter 2, however, the theoretical basis of Seskin's estimates appears to be weak. As a consequence, an explicit comparison can be made between his estimates and those presented here only with great difficulty if at all. Nevertheless, the results are consistent with the prediction made by Harrington and Portney (1983), that the compensating variation approach should give higher willingness to pay values than the cost of illness approach.

There are at least two reasons, however, why the willingness to pay estimates presented in Table 5 are comparatively low. First, the reductions in ozone levels contemplated are not large. As indicated, the mean of the variable OZONEM was .019 ppm, reflecting the fact that ozone levels in St. Louis are much lower than those elsewhere in the U.S. In the Los Angeles area, for example, average ozone concentrations would exceed that figure by a factor of more than five and peak ozone concentrations would be as high as .35 ppm. In any event, for the case of St. Louis, a 10 percent or even a 30 percent reduction in ozone levels is not particularly large in absolute terms. Second, the benefit estimates account only for the affects of improvements in air quality on health. A total benefit estimate for each individual might also account, for example, for reduced materials damage and improved visibility.

5. <u>Conclusion</u>

In this chapter, the empirical application of the theoretical model was presented and estimates of willingness to pay for the "average" employed person were derived for specific reductions in air pollution. A method was derived whereby the health stock of an individual did not need to be quantified. This allowed for possibly more reliable results as no valid measure of health stock could be found. It appears from the estimations of the implicit health production function that ozone levels do play an important role in the production of health. No other pollutants were shown to have as large of effect on the production of health as ozone. For this reason only willingness to pay estimates were made for a reduction in ozone. Those willingness to pay estimates reflected an annual per employed person benefit to St. Louis residents of between $2.42 and $2.69 for a 10 percent reduction in mean ozone levels and between $7.27 and $8.08 for a 30 percent reduction in mean ozone levels.

FOOTNOTES

1. Recall that in the 2197 observation sample, measures of the hourly wage were not available for most respondents. To circumvent this problem in the reduced form regressions, the arithmetic mean of HWAGE was substituted in place of the missing and unknown values. A detailed evaluation of this procedure is provided in a series of four papers by Afifi and Elashoff (1966, 1967, 1969a, 1969b).

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APPENDIX 1

HEALTH EFFECTS OF OZONE ON HUMANS

1. Introduction

     Ozone is one of several photochemical oxidants that can be observed in
the atmosphere. It is a secondary pollutant; that is, it is formed as a
result of chemical reactions involving other pollutants in the atmosphere
and sunlight. (Primary pollutants are those emitted directly by pollution
sources.) The pollutants most responsible for the formation of ozone are
the nitrogen oxides and hydrocarbons. The internal combustion engine is a
major source of these pollutants, although there are other stationary
sources such as electric power generating plants which contribute heavily
to emission of nitrogen oxides.

     The amount of ozone found in the atmosphere will depend upon the time
of day, meteorologic conditions, and the amount of nitrogen oxides present.
Early in the morning the concentration is low. As the day progresses, the
amount of ozone in the air increases. The primary influence is that of
sunlight intensity. Also, rush hour traffic will cause ozone levels to rise
dramatically. The highest concentrations of ozone are usually found between
the hours of 10 a.m. and 6 p.m.

     The concentration of ozone in the atmosphere also will vary widely
with location. Table 1 illustrates this diversity. During the years
1964-1967, St. Louis had a maximal hourly average of .35 ppm with a peak
concentration of .85 ppm while Chicago had a maximal hourly average of only
.13 ppm and a peak concentration only slightly higher at .19 ppm. Los
Angeles had a higher maximal hourly average than St. Louis during this
period of time, .58 ppm, but the peak concentration only reached .65 ppm
(NAS 1977). Ozone concentrations will even vary widely within an SMSA.
Table 2 presents a summary of ozone levels as measured by the RAMS network
in the St. Louis SMSA during the years 1974 to 1977. RAMS station 15
exceeded a maximal hourly average of .01 ppm on 64.68 percent of the total
days with available data and a maximal average of .08 ppm on 9.79 percent
of the total days. RAMS station 17 exceeded a maximal hourly average of
.01 ppm on 75.36 percent of the total days but on only 2.87 percent of the
total days was a maximal hourly average of .08 ppm exceeded. This
variation is due for the most part to the amount of traffic and the
presence of stationary pollution sources in the area.

     It has been estimated that many major U.S. cities will not attain the
ambient air standard for ozone set by the EPA in 1979. The national
primary and secondary standard for ozone is an hourly average of .12 ppm
not to be exceeded more than once a year. Referring to Table 2 and

TABLE 1

SUMMARY OF MAXIMAL OXIDANT CONCENTRATIONS IN SELECTED CITIES, 1964-1967[a]

Station	Total Days With Available Data	Total Days with Maximal Hourly Average ≥ Concentration Specified						Maximal Hourly Avg.(ppm)	Peak Concentration (ppm)	Yearly Average (ppm)
		.05 ppm		.10 ppm		.15 ppm				
		No. of Days	% of Days	No. of Days	% of Days	No. of Days	% of Days			
Los Angeles	730	540	74.0	354	48.5	220	30.1	.58	.65	.030
Denver	285	226	79.3	51	17.9	14	4.9	.25	.31	.036
St. Louis, MO	582	362	62.2	59	10.1	14	2.4	.35	.85	,031
Philadelphia	556	233	41.9	60	10.9	13	2.3	.21	.25	.026
Cincinnati	613	319	52.0	55	9.0	IO	1.6	.26	.32	,030
Washington, D.C.	577	313	54.2	65	11.3	7	1.2	.21	.24	.029
Chicago	530	269	50.8	24	4.5	0	0	.13	.19	.028

[a]From NAS 1977 (derived from U.S. DHEW)

TABLE 2

SUMMARY OF OXIDANT CONCENTRATION IN ST. LOUIS, 1974-1977 (RAMS AIR POLLUTION DATA)

Station	Total Days With Available Data	.01 ppm		.05 ppm		.08 ppm		.12 ppm		.16 ppm	
		No. of Days	% of Days	No. of Days	% of Days	No. of Days	% of Days	No. of Days	% of Days	No. of Days	% of Days
1	420	303	72.14	132	31.43	46	10.95	9	2.14	2	.5
2	438	338	77.17	129	29.45	40	9.13	10	2.28	1	.2
3	428	283	66.12	1 04	24.30	26	6.07	5	1.17	2	.47
4	424	279	65.80	78	18.40	22	5.19	4	.94	0	0
5	431	295	68.45	102	23.67	28	6.50	4	.93	1	.23
6	421	303	71.97	129	30.64	39	9.26	10	2.38	5	1.19
7	434	288	66.36	87	20.05	27	6.22	3	.69	2	.46
6	433	323	74.60	124	28.64	39	6.24	10	.69	2	.116
9	4 20	300	71.43	178	42.38	65	15.48	12	2.86	1	.48
10	427	328	78.10	150	35.13	42	9.84	9	2.10	0	0
11	428	323	75.47	123	28.74	39	9.11	10	2.34	2	.47
12	428	316	73.83	112	26.17	32	7.48	10	2.34	3	.70
13	432	324	75.00	121	28.01	37	8.56	7	1.62	3	.69
14	421	316	75.06	158	37.53	52	12.35	17	4.04	5	1.18
15	419	271	64.68	113	26.97	41	9.79	10	2.39	2	.46
17	418	315	75.36	108	25.84	12	2.87	0	0	0	0
19	422	301	71.33	147	34.83	53	12.56	13	3.08	4	.95
20	402	270	67.16	108	26.87	34	8.46	10	2.49	0	0
21	422	319	75.59	154	36.49	54	12.80	16	3.79	6	1.42
Average % of Days St, Louis Exceeded Stated Concentration			71.87		29.24		8.89		2.01		.52

Total Days With Maximal Hourly Average > Concentration Specified

Average Ozone Level   .019
(1974-1977)

averaging across stations, St. Louis exceeded an hourly average of .12 ppm
on 2.01 percent of the days with available data. This translates into
nearly eight days a year where an hourly average of .12 ppm was exceeded;
thus, during the years 1974-1977, St. Louis was in violation of the present
air quality standards for ozone.

It is interesting to note, however, the decline in ozone levels that
St. Louis experienced from 1967 to 1975. From 1964 to 1967, the maximal
hourly average exceeded .05 ppm on 62.2 percent of the total days while
from 1974-1977 this happened on only 29.24 percent of the total days. The
average ozone level declined from .031 ppm during 1964 to 1967 to .019 ppm
during 1974 to 1977.

2. Health Effects on Humans

Two types of studies have been conducted to determine the health
effects of ozone on humans. These are controlled experimental studies
under laboratory conditions and epidemiological investigations of
ozone-exposed groups. From these studies, ozone has been found to produce
adverse effects on pulmonary function, numerous discomforts (including
headaches, irritation and soreness of the throat, nose, mouth, and trachea,
substernal discomfort, coughing, wheezing, and malaise), changes in blood
chemistry, and possibly chromosomal aberrations.

A. Pulmonary Function and Sensory Irritation

1) Controlled experimental studies

Controlled experimental studies of the health effects of ozone on
humans offer several advantages (Hallenbeck et al. 1979). These
experiments can be carried out under specific conditions. That is, many
factors such as temperature, humidity, health status of the participants,
duration of exposure, and level of exercise can be controlled for and thus,
the effects that are produced can be directly related to the pollutant,
ozone. Also, there are several pulmonary tests that can be carried out in
the laboratory so that specific effects of ozone on the lungs can be
identified.

Ozone concentrations of 4-30 ppm have been found to cause death in
laboratory animals after exposure at these levels for 3-4 hours.
Industrial exposure at 9, ppm has produced severe pneumonia in workers
(Young et al. 1964). Ozone levels below 1 ppm are the most interesting to
study though because they are known to occur in industries, smog, and in
the cabins of modern aircraft. Pulmonary function and sensory irritation
have been studied most frequently for levels of ozone exceeding .30 ppm.
Very few studies have been conducted for ozone levels of .20 ppm, .10 ppm,
or below. Those studies that have been carried out at these levels have
found no conclusive evidence of adverse effects (Equitable Environmental
Health, Inc. 1977). Referring back to Table 2, St. Louis never experienced
an hourly average ozone level of .30 ppm during the years 1974 to 1977 and

an hourly average of .16 ppm was exceeded on only .52 percent of the total days monitored.

Decrements in pulmonary function have been reported for ozone levels greater than .30 ppm. Along with these changes in pulmonary function many symptoms of sensory irritation have been reported. Below is a review of several of these studies.

Bates (1972) exposed 10 healthy subjects to .75 ppm of ozone for two hours while the subjects were resting. Symptoms reported by the subjects included substernal soreness and cough. A few complained of symptoms of dyspnea (difficulty in breathing) and pharyngitis (inflammation of the pharynx). The pulmonary tests showed decrements in vital capacity (maximal expiration following maximal inspiration), increased airway resistance, and decreased dynamic compliance of the lungs (greater stiffness of the lungs). The researchers concluded that .75 ppm of ozone, in absence of other pollutants, is too high a level for a two hour exposure of the general population. Folinsbee et al. (1975) reported a decrease in maximum oxygen uptake (absorption) following exposure to ozone at .75 ppm with intermittent exercise. Because of this decrease in maximum oxygen uptake, maximum work performance was reduced 11 percent. Kagawa and Toyama (1975) found that peak concentration of .90 ppm of ozone for 5 minutes produced adverse effects when the subject undertakes exercise in it. They found a highly significant decrease in airway conductance in the four subjects who were exposed though none of the subjects complained of any symptoms.

In an earlier study, before researchers used environmental chambers to simulate ambient air with ozone pollution, eleven subjects breathed .60-.80 ppm of ozone through a mouthpiece for two hours (Mohler et al. 1970). This level of exposure produced decrements in vital capacity, forced expiratory volume, and other pulmonary functions. Subjects complained of substernal soreness and tracheal irritation for 6 to 12 hours after the exposure. These symptoms were accompanied by a dry cough. The effects on pulmonary function and the symptoms experienced could be partially due to the way the subjects were exposed, i.e. through a mouthpiece.

Other studies have produced similar results to those studies mentioned (see EPA 1974). It appears that there are definite physiological changes that take place in the lungs and these are accompanied by various symptoms when subjects are exposed to .75 ppm for two or more hours.

Experimental exposures at .50 ppm have generally had similar effects on pulmonary function as exposure at .75 ppm. Hackney et al. (1975a) exposed 2 groups of 4 subjects each to ozone levels of .50 ppm for 4 hours. Subjects in Group A were healthy with no history of pulmonary problems while subjects in Group B had a history of hyperreactive airways (they had frequently suffered from allergies, smog sensitivity, or asthma). Group A showed few or no effects; they complained only of mild pharyngitis and substernal discomfort. Group B, however, developed marked respiratory symptoms and physiological changes. Pulmonary tests revealed decreases in lung capacity, forced expiratory flow rates and volume and forced vital capacity, reductions in inspiratory capacity due to substernal pain, and

increased airway resistance. Symptoms in Group B included substernal pain, wheezing, malaise, and cough. The authors concluded that subjects with a history of pulmonary hyperactivity were significantly affected while healthy subjects were minimally affected by exposure at .50 ppm. However, subsequent exposure of a second group of 7 healthy subjects at .50 ppm ozone 2 hours a day for 2 consecutive days with intermittent exercise resulted in definite decreases in pulmonary function (1975b). Subjects showed symptoms of cough. substernal discomfort, and malaise. The effects were greatest on the second consecutive day of exposure. This finding is consistent with the results of other studies that have found effects of ozone on pulmonary function to be more severe during exercise. In these same experiments, some effects were found in the same group at exposure levels of .37 ppm but not after exposure at .25 ppm. There were definite decreases in pulmonary function and various symptoms were observed at .37 ppm.

In a study by Folinsbee et al. (1977), 14 nonsmoking males were exposed to .50 ppm ozone for 2 hours under different environmental conditions. The subjects were divided into 2 groups; one that performed from 60 to 90 minutes of exercise and one that performed 30 to 60 minutes. The temperature of the laboratory chamber was also varied. The authors found that pulmonary function changed the greatest amount immediately after exercise instead of at the end of the exposure. The decrease in forced volume capacity and forced expiratory volume following exercise was twice as large as the decrements at the end of exposure. The greatest decrease occurred when heat and ozone were combined. The symptoms observed in the subjects were chest discomfort and difficulty in taking a breath. The authors concluded that effects of ozone are most severe immediately after exercise, and heat stress may modify the overall effect of ozone on pulmonary function.

Several other studies have also found pulmonary function is affected at .50 ppm ozone. Kerr et al. (1975) found significant changes from control values of several pulmonary tests (airway conductance, pulmonary resistance, and forced expiratory volume) when 20 subjects were exposed to .50 ppm ozone for 6 hours with two intermittent exercise sessions of 15 minutes each. Folinsbee et al. (1975) studied the response of 28 subjects after ozone exposure of .50 ppm for 2 hours while exercising intermittently at 45, 60, and 75 percent of maximum aerobic power. The major response noted was an increase in respiratory frequency during exercise following ozone exposure. It was concluded that through its irritant properties ozone modifies the normal ventilatory response to exercise.

There is also evidence that ozone can produce adverse effects on pulmonary function and discomfort at concentrations below .50 ppm. One study by Hackney et al. (1975b) that examined effects of ozone exposure of .37 ppm has already been noted. The authors found definite decreases in pulmonary function, and the subjects experienced various discomforts. Hazucha and Bates (1975) reported a marked decrease in pulmonary function among healthy subjects performing light exercise while exposed to .37 ppm of ozone and .37 ppm sulfur dioxide for 2 hours. Throat irritation, coughing, and chest pain were also observed. These effects persisted

several hours after exposure. Folinsbee et al. (1975) noted an increase in respiratory frequency when subjects were exposed to .37 ppm ozone for 2 hours while exercising intermittently.

Effects were studied by DeLucia and Adams (1977) on 6 males after exposure for 1 hour to .15 ppm or .30 ppm ozone at rest or performing exercise at 25, 45, or 65 percent of maximum oxygen uptake. Ventilation volume and maximum oxygen uptake were unaffected by the most severe exposures and exercise. Host subjects did however demonstrate signs of toxicity (headaches, congestion, and wheezing) during the most stressful protocols. Two of the most sensitive individuals were unable to complete 1 hour of 65% maximum oxygen uptake exercise while breathing .30 ppm ozone.

In most of these studies, an association between symptoms and changes in lung function was usually found. Hypersusceptibles do not develop symptoms upon 2 hour exposure to ozone at .20-.25 ppm. However, symptoms develop in normal as well as hypersusceptibles after exposure for 2 or more hours at levels greater than .37 ppm with or without exercise. Exposure to .15 or .30 ppm ozone for one hour, accompanied by stressful exercise was sufficient to produce discomfort in even normal subjects. The symptoms that subjects experienced most frequently were substernal pain, coughing and wheezing, chest tightness, sore throat, and tracheal irritation. At lower levels of exposure, headaches were frequently complained of. Some hypersusceptible subjects were unable to perform normal tasks or complete the study when exposed to levels from .37 ppm ozone while exercising.

The experimental studies described above indicate that significant adverse effects on pulmonary function occur in humans at ozone concentrations as low as .37 ppm. There is evidence that hypersusceptibles (those with a history of respiratory problems) may develop symptoms at levels lower than this. In many of these studies, subjects engaged in some form of exercise to test the response of ozone on exercising individuals. These studies concluded that the effects of ozone concentrations at any level are much more pronounced when an individual is engaging in "heavy work." The effects of ozone are thus related to the volume of ozone breathed per unit. The more air an individual is breathing in the higher the actual exposure to ozone will be.

Several researchers have reported an apparent adaptation to pulmonary effects of repeated or chronic exposure to ozone. In a recent study by Hackney et al. (1977) 6 subjects were exposed to .50 ppm ozone 2 hours a day for 4 successive days under conditions simulating ambient pollution exposures. Five of these subjects showed pulmonary function decrement on exposure day 1 to day 3. This decrement was largely reversed on day 4. These results suggest some people do not continue to experience the same decrements in lung function after repeated exposures to ozone.

Another study by Hackney et al. (1977) compared the responses of 4 Canadians (whose previous total ambient ozone exposure was low) and those of 4 southern Californians to ozone exposures of .37 ppm for two hours with light exercise. The Canadians, on average, showed greater clinical and physiological reactivity to exposure. The explanation that seemed most

plausible to the investigators was that southern Californians have adapted to chronic ambient ozone exposure.

The purpose of a study by Folinsbee et al. (1980) was to determine whether there was any cumulative effects of repeated exposure to ozone and if adaptation to repeated exposures occurred. Three groups of subjects were used, each exposed to a different concentration of ozone: group 1 (n=10) was exposed to .20 ppm ozone, group 2 (n = 10) was exposed to .35 ppm ozone, and group 3 (n=8) was exposed to .50 ppm ozone. Subjects were exposed for 2 hours to filtered air on day 1 and day 5 of the experiment and to the ozone air for 2 hours on day 2 through day 4. No acute or cumulative effects were seen for those repeatedly exposed to .20 ppm ozone. There were decrements in pulmonary function for those exposed to .35 ppm on day 2 and 3, the greatest effect being seen day 3, but the effects were absent on days 4 and 5. The greatest change in pulmonary function came from the group repeatedly exposed to .50 ppm ozone. The most significant effects occurred day 3. There were significant effects day 4 but they were of a lesser magnitude. The authors concluded that there are some short term cumulative effects. These are followed by a period of resistance or adaption in which there is a marked lessening of the effects on pulmonary function and of subjective symptoms.

Synergistic effects of ozone and other air pollutants have been reported. In a study by Hazucha and Bates (1975), healthy young subjects were continuously exposed over a 2 hour period to a mixture of .37 ppm ozone and .37 ppm sulfur dioxide. They found that the combination of these two pollutants had a much greater effect on pulmonary function than either had individually. Greater decrements in pulmonary tests were observed following the combined exposure than was observed following exposure to each gas alone. However, a study that was designed to confirm this conclusion, using the same levels of ozone and sulfur dioxide found a substantially smaller decrement in pulmonary function than what had been previously reported.

Hackney et al. (1975a) exposed subjects to a mixture of ozone and nitrogen dioxide and ozone, nitrogen dioxide, and carbon monoxide. Ozone concentrations measured .25 ppm and .50 ppm. Added to these concentrations was .30 ppm nitrogen dioxide or .30 ppm nitrogen dioxide and 30 ppm carbon monoxide. The authors concluded that no additional effects were produced nor were effects more severe due to the increase of nitrogen dioxide and carbon monoxide. However, no conclusions about synergistic effects can be drawn from this study because of the possibility of these effects being present at different pollutant levels or environmental conditions.

2) Epidemiological Studies

A variety of epidemiological studies attempting to associate various characteristics of human health with daily concentrations of ozone have been carried out in the last two decades, mainly in the Los Angeles area. Many of the epidemiological studies of the health effects of ozone on human beings suffer from serious defects. Among these are lack of information about subjects studied, subjectivity of response due to knowledge of the

level of exposure, lack of analysis of the threshhold of the response, incomplete separation of the effects on health of competing meteorological and pollutant variables, and lack of appropriate weighting of time spent indoors and outdoors and the associated differences in pollutant exposure Hallenbeck 1979). Within these limits, some definite conclusions can be drawn as to the association of some health indicators with levels of ozone,

A relationship between mortality and concentrations of ozone has not been clearly demonstrated. Two studies relating the effects of ozone on the mortality of the elderly in Los Angeles County have been reported by the California State Health Department (EPA 1974). A marked increase in mortality among the elderly was found during a period of high photcchemical-oxidant concentrations during a 2 week period. However, there was also a sharp increase in temperature during this period that contributed to the increase in mortality. When mortality rates during high oxidant concentrations accompanied by low temperatures were examined, no relationship between oxidant levels and mortality could be discerned. The other study compared daily mortality and transfer to a hospital among nursing home residents to daily temperatures and the occurrence of smog-alert days with ozone levels of .30 ppm or higher. No correlation between mortality and smog-alert days in the absence of high temperatures could be found.

Sterling et al. (1966) studied the influence of ozone on the number of people admitted to hospitals for a 7 month period in 1961. They grouped diseases into "highly relevant," "relevant", and "irrelevant" categories. Highly relevant diseases were allergic disorders, inflammatory diseases of the eye, acute upper respiratory infections, influenza, and bronchitis. "Relevant" disorders included disease of the heart, rheumatic fever, vascular diseases, and all other respiratory diseases. All other disorders were considered irrelevant. Correlation coefficients between admissions for "highly relevant" diseases and nine pollution indices measured were all statistically significant at the .05 level. Correlations between "relevant" diseases and pollution were significant for oxidant, ozone, and sulfur dioxide. For all pollutants except sulfur dioxide, correlations between irrelevant diseases and pollution were negative cr not significant. This study must be interpreted carefully; the relationships, though significant, were minimal. Confirmation of this study over a longer period is needed.

Several studies have also found some evidence that ozone may aggravate existing respiratory conditions. Schoettlin and Landau (1956) studied 137 patients being treated for asthma in the Pasadena area. No correlation between air pollution of any kind and asthmatic attacks was significant. However, a significantly greater number of asthma attacks occurred on days when ozone levels were greater than .25 ppm than on days when they were less than .25 ppm. This result may suggest that there is a threshold level of ozone above which an increase in asthmatic attacks may occur.

Studies were done by Motley et al. (1959) on 66 volunteers, 46 of whom had pulmonary emphysema. Oxidants were removed from the air of the laboratory room by activated charcoal filters. Pulmonary functions

improved in the volunteers with emphysema if they entered the room on a "smoggy" day and remained there for 40 hours or more. No change in pulmonary function was seen for normal subjects or patients with emphysema who entered the room on a "nonsmoggy" day. The smoking habits of subjects were not accounted for nor were the effects of individual pollutants separated from one another so no real conclusions can be drawn.

Other studies have suggested an association between exposure to ozone and both increased frequency of asthma attacks and decrements in pulmonary function (EPA 1974). However all of these studies have serious limitations. Variations in smoking habits haven't been taken into account nor have the effects of other pollutants either singly or in combination with ozone been controlled for.

Studies have also been done to examine the effects ozone may have on the healthy population. Hammer et al. (1974) studied the effects of ozone levels on student nurses in Los Angeles for the period October 1961 to June 1964. Diaries were kept by student nurses on any discomfort or illness they had during each day of the study period. Headache frequency rose slightly at and above ozone levels of .25-.29 ppm. Rates for eye discomfort began to increase at .15-.19 ppm; rates for cough and chest discomfort showed an increase at .30-.39 ppm. Symptom frequencies in this study were more closely related to photochemical oxidants than carbon monoxide, nitrogen dioxide, or daily minimum temperatures.

Effects of ozone on the health of college students in the Los Angeles and San Francisco Bay area were examined by Durham (1974). Health data were collected from 1970-71. Each time a student visited the health service for a new illness, a sheet requesting demographic information, smoking habits, and physicians' diagnosis was completed. Coefficients of correlation between levels of pollution and weather variables on day 1 and proportions of new illness on the same day and on days up through day 7 were made. The illnesses most strongly associated with pollution were, in descending order, pharyngitis, bronchitis, tonsillitis, colds and sore throat. The pollutants most closely correlated with illness were in descending order, peak oxidant, mean sulfur dioxide, mean nitrogen dioxide, and mean nitric oxide. Stronger associations between pollution and illness were found in the Los Angeles area. Correlations of pollution with bronchitis were greatest when lagged 5 to 6 days; correlations of pollution with combined respiratory disease were greatest when lagged zero to 3 days; and correlations of pollution with asthma, eye irritation, headache, and hay fever were greatest when lagged zero to 1 day. This study strongly suggests a relationship between ozone levels and specific types of illnesses.

Pearlman et al. (1971) studied the effects of oxidants upon epidemic influenza incidence and duration of the illness. During 1968-69, morbidity rates of 3500 children from 5 southern Californian communities representing a definite gradient in chronic ozone exposure were studied to see if there was a relationship between chronic ozone exposure and epidemics among school children. Information from several sources (absenteeism from school

111

and questionnaries filled out by parents) revealed no statistically significant morbidity differences corresponding to ozone exposure.

Effects of Los Angeles oxidizing type air pollution on athletic performance was examined by Wayne et al. (1967) in 21 competitive meets of high school cross country track runners from 1959-64. It was hypothesized that running times should improve as the season progresses. Team performance was evaluated by the percentage of boys who didn't improve over their last meet. The highest correlation to team performance was that of oxidant levels in the hour before the race. Carbon monoxide levels, temperature, or humidity showed no relationship with team performance. The authors asserted that the decrease in performance may be directly physiological or may be due to decreased motivation caused by discomfort It is noted that athletes often complain of chest discomfort after competing in Los Angeles. Whether or not the decrement in team performance is due to physiological changes or decreased motivation, the study still indicates that ozone had an effect on team performance.

Though no conclusive evidence can be drawn from these studies, there is still an indication that low levels of ozone result in adverse human responses. Many participants suffered from discomfort when the concentration of ozone in the atmosphere was as low as .15 ppm. Symptoms reported at this exposure included eye irritation, sore throat, shortness of breath, cough, headache, watering of eyes, and hoarseness. Asthmatic attacks increased when ozone concentrations were above .25 ppm. At .3-.39 ppm, chest discomfort was reported by participants in these studies. Therefore, it seems safe to say that members of the general population suffer discomfort at ozone levels that are found in urban areas.

B. Effects on Blood Chemistry

At almost every level of ozone above .2-.25 ppm, small but significant changes in human blood biochemistry are seen. The changes include increases in red blood cell membrane fragility and serum E levels, decreases in hemoglobin concentrations, and alterations in the activities of several enzymes in the blood. Although there is a wide spectrum of ozone interference on blood biochemistry, the physiological Significance is unknown.

C. Mutagenesis

Ozone may potentiate chromosomal aberrations. Several studies have been done to evaluate these possible effects. Merz et al. (1975) studied the effects of exposure to .50 ppm. ozone for 6 to 10 hours on chromosomal aberrations. Ho true chromosome type aberrations were found. However, chromatid deletions and single strand breaks increased after exposure. The authors reported an increased frequency in these abnormalities 2 weeks after exposure with a return to normal frequency 6 weeks after exposure.

Another study (Fetner 1965) found chromatid deletions were produced as an exponential function, of the exposure to ozone. The authors concluded

that exposure to ozone is capable of producing chromatid breakages in human cell cultures.

3. Conclusion

Conclusive evidence exists that high ozone levels are detrimental to human health. Very pronounced physiological effects of ozone on pulmonary function have been found for ozone levels of .50 ppm or higher. Less pronounced effects have occurred in studies where the level of ozone administered to subjects was .37 ppm with these effects becoming more pronounced when subjects were engaged in heavy work. Sensory irritation and discomfort have been reported at ozone levels as low as .15 ppm, a level not uncommon in many urban areas.

The next chapter summarizes the relationships found by researchers between levels of lead in the body and the resulting physiological effects. WHile it is a fairly direct procedure to find a relationship between the level of ozone in the air and its physiological effects, it is much more difficult to compare actual lead concentrations in the ambient air to the effects this concentration may have on health. Lead occurs in media other than the ambient air, and the contributions each media has on an individual's total exposure to and absorption of lead is not clearly known. Therefore, it will be an impossible task to relate concentration so flead only in the ambient air to specific physiological effects; only the total lead concentration found in the subjects' bodies can be used to find a relationship between lead levels and health.

APPENDIX 2

HUMAN HEALTH EFFECTS OF LEAD

1. Introduction

     Every member of the general population is exposed to elevated levels
of lead from numerous sources. It is found in the air, water, foods,
tobacco, soil, dust, and other items. The exposure to lead will depend
upon both how much is found in these different medium and the amount
actually inhaled or ingested. Some groups of people may be subject to much
higher exposures than others. For example, children may eat soil or dust
that contain lead deposited from the air or paint on walls. Also, some
workers are exposed to large amounts of lead due to the type of occupation
they are in.

     Exposure to lead for the general adult population may be summarized as
follows. Average inhalation from the air is about 15 $\mu g/day$ in urban
areas. Inhalation may be as low as 1 $\mu g/day$ in rural areas to more than
100 $\mu g/day$ at some urban sites. Average ingestion from diet is about
200 to 300 $\mu g/day$, with a range anywhere from 100 μg to more than 2000
$\mu g/day$. Ingestion from water ranges from 1 to more than 500 $\mu g/day$.
About 10 μg of lead per pack of cigarettes smoke is inhaled. Ingestion
of lead from dust, soil, or paint is negligible in adults; however,
ingestion may be much higher in children.

     It is clear from this data that diet is the major pathway of exposure.
However, no conclusions about actual body burden can be made until it is
known how much of this inhaled or ingested lead is absorbed. Also, it is
clear that typical exposure can include a wide range of values. Urban
residents or smokers will be exposed to more lead than rural residents or
nonsmokers. Other characteristics of individuals such as age, sex, and
socioeconomic status will affect the degree of lead exposure and the amount
absorbed.

     The relationship between exposure to lead and the level of lead in the
body has not been clearly defined. The EPA (1977) estimated that of the
lead in the air inhaled, between 20 and 40 percent of it would be deposited
in the lungs. It is not known how much of the deposited lead is absorbed
into the body. From controlled studies, it has been estimated by the EPA
(1977) that for every 1 $\mu g/m$ in the air, the blood lead level in an
individual increases by approximately 1 to 2 $\mu g/100$ ml. The amount
absorbed from the gastrointestinal tract is about 8 to 10% of the amount
ingested. The EPA estimated that for every 100 $\mu g/day$ of oral intake of
lead an increase in blood lead levels of between 4.4 to 18.3 $\mu g/100$ ml
takes place. The amount of lead absorbed into the body from inhaled

cigarette smoke is not known though it is assumed to be about the same amount as that inhaled from the air.

The relative contributions of each medium to the total lead body burden is also important in considering lead exposure. The contributions will vary widely in accordance to the residence of an individual and the characteristics of that individual. For example, an urban smoker will get a much higher percentage of lead from the air and tobacco smoke while a rural nonsmoker will get very little from these sources. In both cases, diet will probably contribute over 50 percent of the total lead absorbed, but for the rural nonsmoker the contribution will probably be about 90 percent while the contribution to the urban smoker will be about 50 percent. Air and tobacco smoke may account for approximately 15 percent each of the lead absorbed into the body of the urban smoker depending of course on the concentration of lead in the air and the number of cigarettes smoked. For the rural nonsmoker, the contribution of these two medium to lead in the body is negligible.

It is difficult to estimate a typical blood lead level for the general population. The differences in estimated blood lead levels are influenced by the degree and type of exposure, the characteristics of the individuals themselves, and the method of sampling and analytical techniques used in the estimation. The average blood lead level of urban adults has been estimated to be between 20 and 25 $\mu g/100$ ml by many researchers. Other studies have found both lower and higher blood lead levels in urban adults. The range of values of blood lead levels for the general population is probably from 10 $\mu g/100$ ml to 30 $\mu g/100$ ml.

A.   Ambient Air Exposures of Lead in the Recent Past

Since 1957, samples of particulate matter collected at many urban and nonurban National Air Surveillance Network (NASN) sites have been analyzed for lead. However, only since 1966 have reliable procedures for measuring lead levels been used; therefore, only this data has been thoroughly analyzed. This data from NASN was studied for trends over the ten-year preiod from 1965 through 1974. It was found that urban lead concentrations increased from 1965 to 1971 and then declined. The principle reason for the decline was the introduction of automobile engines around 1970 that used gasoline with lower lead content. Practically all cars built after 1970 were able to use regular gasoline instead of the more leaded premium fuels. Even lower levels of lead in the air are probably now present because of the large use of unleaded gasolines. This decline will continue as more people switch from cars that use regular gasoline to cars that use unleaded gasoline.

In Table 1, lead concentrations and particle size are presented for the four quarters of 1970 for six urban areas. The average annual total lead concentration ranged from a high quarter of 3.2 $\mu g/m^3$ in Chicago to a low quarter of 1.3 $\mu g/m^3$ in Washington, D.C. St. Louis experienced a high quarter of 1.9 $\mu g/m^3$ and a low of 1.6 $\mu g/m^3$. This table indicates tghat many U.S. urban areas in 1970 were exceeding the EPA standard of 1.5 $\mu g/m^3$ maximum arithmetic mean average over a calendar quarter. A

115

TABLE 1

QUARTERLY AND ANNUAL SIZE DISTRIBUTIONS OF
LEAD-BEARING PARTICLES FOR SIX CITIES, 1970[a]

City and Quarter of Year	Average Concentration $\mu g/m^3$	Average Mass Media Diameter μ_m	Percentage of Particles $\leq \mu_m$
Chicago, IL			
1	2.4	1.43	41
2	3.5	.51	65
3	3.5	.56	65
4	2.9	.54	64
Total Year	3.2	.68	59
Cincinnati, OH			
1	1.0	.25	79
2	2.2	.41	74
3	1.9	.54	69
4	2.1	.65	67
Total Year	1.8	.48	72
Denver, CO			
1	2.0	.43	76
2	1.1	.58	68
3	1.4	.52	69
4	3.0	.56	66
Total Year	1.8	.50	70
Philadelphia, PA			
1	1.5	.36	74
2	1.2	.38	74
3	1.8	.70	62
4	1.9	.45	70
Total Year	1.6	.47	70
St. Louis, MO			
1	1.9	.46	68
2	1.6	.63	53
3	1.8	.78	59
4	1.8	.95	53
Total Year	1.8	.69	62
Washington, D.C.			
1	1.3	.36	76
2	1.0	.39	73
3	1.3	.41	74
4	1.8	.54	71
Total Year	1.3	.42	74

Source: EPA (1977)

large number of southern California cities had annual average concentrations of 3.0 $\mu g/m^3$ or greater during this period. This was probably due to the heavy automobile traffic in these areas and also the topography and meteorological condition sthat favor retention of pollutants.

The average max median diameter for these six cities ranged from .69 mm in St. Louis to .42 mm in Washington, D.C. Forty-nine to 74 percent of the lead was associated with particles less than 1 mm in diameter. The smaller particles can become embedded in the lungs and therefore cause more health problems than the larger particles.

Table 2 presents a summary of lead concentrations in the St. Louis metropolitan area for the five quarters in the period January , 1976 to March, 1977. When these figures are compared with those in Table 1, the amount of lead present in the ambient air appears to have decline significantly since 1970. In fact, the maximum arithmetic mean over one quarter in 1976 is 1.040 $\mu g/m^3$ compared to 1.9 $\mu g/m^3$ in 1970. A large part of this reduction is probably due to the increased use of unleaded gasoline in automobiles. From this data, it can be concluded that St. Louis was meeting the EPA standards for lead in the ambient air in the period when the RAMS data was gathered. The level of lead in the ambient air declined approximately 100 percent over the period 1970 to 1976 therefore bringing St. Louis in compliance with the primary and secondary EPA standards.

It is difficult to know the contribution this level of lead in the ambient air has on the total lead burden in an individual. As mentioned in the previous section, the EPA (1977) has estimated that for every 1 $\mu g/m^3$ in the air, the blood level increases by approximately 1 to 2 $\mu g/100$ ml. If this is true, then the amount of lead in the air in St. Louis is probably contributing little to the total level of lead in the bodies of those living in the St. Louis SMSA. The health effects derived from airborne lead as compared to the lead in other media may actually be quite small.

2. <u>Health Effects of Lead</u>

The toxicity of lead has been recognized since ancient times when workers exposed to lead were observed to suffer more frequently from symptoms of lead encephalopathy. Lead affects many organs and organ systems. The effects of lead on the hematopoietic system (the blood forming system) and the central nervous system and the amount of lead exposure at which these effects occur have been most extensively studied and documented. In addition to the effects lead has on these two systems, lead also has chronic toxic effects on the kidneys, the liver, the skeleton and the gastrointestinal, cardiovascular, endocrine, immune, reproductive, and peripheral (neuromuscular) systems. It is now thought that there may be relationships between lead and chromosomal abnormalities, mutations, and cancer.

117

TABLE 2

QUARTERLY LEAD CONCENTRATIONS IN THE ST. LOUIS AREA, 1976-1977[a] ARITHMETIC MEAN OVER A QUARTER
(VALID CASES IN PARENTHESES)

Station	Quarter 1			Quarter 2			Quarter 3			Quarter 4			Quarter 5		
	Small size[b] $\mu g/m^3$	Large size[c] $\mu g/m^3$	Total $\mu g/m^3$	Small size $\mu g/m^3$	Large size $\mu g/m^3$	Total $\mu g/m^3$	Small size $\mu g/m^3$	Large size $\mu g/m^3$	Total $\mu g/m^3$	Small size $\mu g/m^3$	Large size $\mu g/m^3$	Total $\mu g/m^3$	Small size $\mu g/m^3$	Large size $\mu g/m^3$	Total $\mu g/m^3$
3	.461 (306)	.144 (311)	.605	.462 (336)	.134 (339)	.596	.573 (285)	.142 (276)	.715	.683 (305)	.158 (299)	.841	.421 (240)	.108 (240)	.529
5	.6C0 (357)	.187 (355)	.787	.523 (346)	.167 (345)	.690	.629 (329)	.180 (326)	.809	.813 (259)	.227 (255)	1.040	.610 (247)	.1C8 (246)	.778
6	.6C8 (173)	.207 (173)	.875	.633 (174)	.205 (176)	.839	.690 (105)	.193 (103)	.889	*	*	*	*	*	*
8	.623 (171)	.247 (172)	.870	.512 (166)	.144 (166)	.656	.571 (166)	.143 (166)	.714	.626 (117)	.128 (118)	.754	.496 (111)	.119 (111)	.615
12	.613 (179)	.199 (179)	.812	.617 (170)	.185 (172)	.802	.804 (151)	.199 (153)	1.033	.850 (155)	.146 (152)	.996	.594 (127)	.152 (131)	.746
15	.307 (171)	.071 (169)	.378	.292 (154)	.081 (152)	.373	.329 (216)	.049 (216)	.378	.426 (156)	.064 (154)	.490	.456 (137)	.062 (138)	.518
18	.242 (159)	.086 (159)	.328	.255 (165)	.078 (164)	.333	.284 (134)	.086 (150)	.370	.360 (159)	.102 (158)	.462	.251 (139)	.097 (137)	.348
20	.445 (127)	.108 (132)	.553	.499 (174)	.121 (177)	.620	.596 (147)	.145 (148)	.741	.602 (142)	.129 (142)	.731	.480 (135)	.107 (132)	.587
22	.168 (154)	.033 (152)	.201	.156 (167)	.028 (163)	.184	.193 (132)	.026 (133)	.219	.197 (152)	.033 (143)	.230	.160 (106)	.027 (104)	.187
24	.183 (164)	*	*	.117 (109)	*	*	.174 (134)	*	*	*	*	*	*	*	*

[a] RAMS Air Pollution Data
[b] < 2 micrometers
[c] > 2 micrometers
* No cases

Dates for Quarters:
1: 1/1/76 - 3/31/76
2: 4/1/76 - 6/30/76
3: 7/1/76 - 9/30/76
4: 10/1/76 - 12/31/76
5: 1/1/77 - 3/14/77

A.   <u>Effects on the Nervous System and Behavior</u>

That workers exposed to large amounts of lead suffer more often from encephalopathy has been recognized since the time of Hippocrates. The symptoms observed were dullness, irritability, headaches, loss of memory, and restlessness which often progressed to delirium, coma, convulsions, and even death. These symptoms were also described in infants and young children. The minimum level of lead exposure that results in encephalopathy is still not clearly known. For most adults, lead encephalopathy does not occur until blood lead levels well in excess of 120 μg/100 ml are reached, though there is evidence of acute encephalopathy and death occurring at blood lead levels below 120 μg/100 ml.   For children, the minimum blood lead level required to bring on encephalopathy is probably between 80 and 200 μg/100 ml (EPA 1977). After encephalopathy has occurred and a person has survived, there may be permanent sequelae. It has been found that learning ability is impaired in children who have suffered from lead encephalopathy and in some cases, mental retardation has resulted from the attack (NAS 1980).

Due to the dramatic decrease in the incidence of encephalopathy, a well defined dose-response relationship will probably never be known. Instead the effects of lead exposure at much lower levels than those resulting in encephalopathy have been extensively studied. Those effects that occur at lower exposure levels and the dose required to bring about these effects have been studied mainly for children. Because children are still developing, they are more vulnerable to the effects of lead on the nervous system than adults are.

The concern today is whether children with elevated lead exposure may be experiencing subtle neurological damage without ever exhibiting any of the symptoms of lead encephalopathy. There have been several studies showing higher blood lead levels in mentally retarded children than in control groups. David et al. (1976) compared two groups of mild and borderline mentally retarded children: those whose cause of retardation was known and those whose cause was unknown. The group of children "etiology unknown" had statistically significantly raised blood lead concentrations, and the mentally retarded group with "probable etiology" showed no significant difference in blood lead concentrations from those of the normal control group. They concluded that the association between lead and mental retardation occurs over a much wider range than previously thought, and this association is independent of encephalopathic lead poisoning. In fact, they believe that any rise in lead exposure above 24.5 μg/100 ml must be regarded as potentially noxious.

De la Burde and Choate (1972, 1975) compared the performance on a series of psychologic tests of 4 year old children who had asymptomatic lead exposure between 1 and 3 years of age (blood lead levels ranged from 40 μg/ml to 100 μg/100 ml) to a group of children of the same age and socioeconomic background who presumably did not have significant exposure to lead. They found significant differences on psychologic tests between the 2 groups. Exposed children had significant deficits in global IQ and associative abilities, in visual and fine motor

coordination and in behavior. They then did a follow up study of the same
children at 7 years of age in order to determine whether the differences
detected on the previous tests still existed. They found that the deficits
of the lead exposed children on the psychologic tests were still present
indicating permanent damage done due to lead exposure. They also found
that the behavior disturbances seen in the lead exposed children at 4 years
of age became obvious and more of a problem in the school setting. They
concluded that there is a significant relationship between asymptomatic
lead exposure and deficits in cognitive, perceptual, and behavioral
functioning and thus, there is a need for early detection of subclinical
lead exposure and for adequate preventive measures.

David et al. (1972) compared the incidence of elevated blood lead
levels in five groups of children: (1) a pure hyperactive group with no
cause for hyperactivity (2) a hyperactive group with probable cause
(prematurity) (3) a hyperactive group with possible cause (4) a group
who had recovered from lead poisoning and (5) a nonhyperactive group.
The pure hyperactive group had statistically significant higher blood
lead levels (mean = 26.2 ± 8 µg/100 ml) than the control group (mean =
22.2 ± 9.6 µg/100 ml). The hyperactive children with probable cause did
not show statistically significant differences in blood lead levels from
the control group. While a causal relationship between hyperactivity
and increased exposure to lead can't be proved, the findings of this
study do seem to support the hypothesis that a relationship between
moderate lead exposure and altered motor activity exists.

Another study by Perino and Ernhart (1974) of the same general design
as the study done by de la Burde and Choate concluded that neurobehavioral
deficits do occur in children only moderately exposed to lead. They found
significant deficits on motor functions and behavioral tests given to
preschoolers who had blood lead levels of 40 to 70 µg/100 ml.

In all of these studies, a causal relationship cannot be shown. It
has been asserted that intellectual deficits and hyperactivity could
contribute to excessive lead exposure because of the pica habits of these
children. However, Beattie et al. (1975) found a strong association
between household water lead levels in the homes of pregnant women and
mental retardation of the children born to these women. Blood lead levels
were significantly higher in the retarded children than the control group a
few days after birth. Lead levels of 25.4 ± 12.1 µg/100 ml were measured
in the retarded group and 17.8 ± 4.9 µg/100 ml were measured in the control
group. In this case, the children's high levels of blood lead cannot be
attributed to their behavior. Instead, it can be inferred that lead was a
causal factor in the children's mental retardation.

Most of the epidemiological studies done in this area have serious
limitations and are open to criticism (NAS 1980). Many of the studies deal
with unusual populations of children, such as those in schools for the
retarded so it is uncertain whether their findings can be applied to the
"average" child. Also many studies have not controlled for other relevant
factors involved in child development such as parents IQ and socioeconomic
data. Almost all of the studies have relied upon blood lead levels as a

marker of the degree of lead exposure because blood can be obtained easily and harmlessly. However, the level of lead in the blood is not an exact measure of exposure because of storage and movement of lead in other parts of the body, and it is not a direct indicator of lead levels in tissues and organs. Blood is thought to measure primarily recent exposure to lead. Lead in the blood and soft tissues is turned over rapidly; it stays in these areas about four to six weeks. Therefore, when past exposure to lead is important to the study, as it is in the neurobehavioral studies, a different measure for lead exposure must be used. The lead content of deciduous teeth is believed to be a good indicator of total lead exposure over the life of the tooth. The amount of lead in shed teeth has been found to be highly correlated with blood lead levels.

Recently, Needleman et al. (1979) addressed these methodological problems in a study that attempted to measure the neuropsychologic effects of subclinical exposure of lead by comparing the performance of a high lead group to a low lead group on a battery of tests. The lead levels in deciduous teeth were used as a measure of exposure, and only those children whose tooth lead levels fell below the 10th percentile and above the 90th percentile of the entire sample were used as the control group and study group, respectively. They found deficits in several of the neuropsychological tests given to the high lead group, and these remained even after 39 nonlead variables that might affect learning ability and behavior were taken into account. Also, when the two groups were evaluated by teachers the high lead group scored significantly lower on 9 of the 11 indices of classroom performance.

Adults have passed the developmental stages that make children so vulnerable to lead exposure, but the central nervous system is still sensitive to lead toxicity. Various effects on sensory, psychomotor, and psychological functions have been reported in adults with blood lead levels between 40 and 80 μg/100 ml. In one recent study (Lillis et al. 1977), 55 percent of a group of workers with mean blood lead levels of 56 μg/100 ml reported central nervous system symptoms such as tiredness, sleeplessness, irritability, and headache and 39 percent reported muscle and joint pain.

Peripheral neuropathy (as indicated by slowed reflexes and muscular weakness) and altered sensitivity to pain have been observed in workers whose blood lead levels exceed 50 μg/100 ml. Case histories confirm the occurrence of lead induced neuropathies (as indicated by nerve conduction velocity) and other neurological signs such as tremors and wrist and foot drop at blood lead levels of 60 to 80 μg/100 ml and in some cases as low as 30 μg/100 ml (past exposure may have been higher) (EPA 1977).

In summary, there is sufficient evidence to indicate that neurological and behavioral effects do occur at exposures to lead less than those which cause lead encephalopathy. The minimum exposure level or the length of the exposure required is not clearly defined. There is no conclusive evidence that behavior or intellectual development is impaired at blood lead levels below 40 to 50 μg/100 ml, but as mentioned before blood lead levels may be

poor markers of exposure. Recent studies relying on lead in the teeth indicate that neurobehavioral impairment may occur at exposures encountered by some urban children.

B. Effects on Heme Synthesis

The effects of lead on the formation of hemoglobin (the protein that transports oxygen from the respiratory system to each cell) have been widely studied. These effects are detectable at lower levels of lead exposure than is the case with any other organ or organ system. Thus the hematopoietic system is termed the "critical effects organ system."

The effects of lead on heme synthesis are understood much better than the effects of lead on globin synthesis. The process of heme synthesis starts from two small building blocks, glycine and succinate. From these two building blocks a complex molecule, protoporphyrin IX, is formed. The synthesis culminates with the insertion of iron at the center of the porphyrin ring. Heme then combines with specific proteins, one of which is globin, forming hemoglobin (see Figure 1).

Lead interferes at several points in the synthesis of heme. The two most important points are the inhibition of the enzyme, δ-aminolevulinie acid dehydratase (ALAD) and the interference in the incorporation of iron into the heme. ALAD is the enzyme that acts to connect two molecules of delta aminolevulinic acid (ALA) to form the porphobilinogen ring. As a . result of this interference in the synthesis, precursors to heme, delta aminolevulinic acid, coproporphyrin (CP) and protoporphyrin IX (PROTO) or free erythrocyte protoporphyrin (FEP) can accumulate to elevated levels in the blood.

Inhibition of ALAD Is reflected in increased levels of its substrate, ALA, in the urine. Inhibition starts at a very low blood lead level. Granick et al. (1973) found that inhibition of ALAD in children started as low as 15 μg/100 ml. Nordman and Hernberg (1975) found a statistically significant correlation between ALAD and blood lead levels not exceeding 10 μg/100 ml. Because the inhibition of the enzyme ALAD is one of the earliest effects of lead on heme synthesis, the amount of ALA in the urine is often used as a detector of lead poisoning. Studies have shown that an increase in blood lead levels bring an exponential increase in ALA urinary excretion (Hernberg et al. 1970; Wado 1976).

When lead interferes with the insertion of iron into protoporphyrin IX, it causes the unutilized protoporphyrin IX to accumulate. The exact method of interference is not clearly known; it may be caused by an inhibition of the enzyme, heme synthetase, or the inhibited entry of iron into the mitochondrian. Large amounts of PROTO can accumulate and occupy the available heme pockets in hemoglobin. It remains incorporated into the hemoglobin molecule for the life of the cell, about 120 days. There is also an accumulation of free erythrocyte protoporphyrin (FEP) in the blood and an accumulation of coproporphyrin, a precursor to PROTO, in the urine. The amount of FEP in the erythrocytes has been used as a sensitive indicator of the amount of

Figure 1

Effects of Lead on Heme Metabolism

(Mitochondrian)

Succinyl

Heme

Glycine

Ferrochelatase

Interference
here

ALA Synthetase

F_e ← F_e

F_e

δ-Aminolevulinic
Acid (ALA)

Protoporphyrin IX

(Cytoplasm)

ALA Dehydrose (ALAD)

Coprophyrinogen III

Inhibition
here

Porphobilinogen

Uroporphyrinogen III

123

lead in the body since the development of a simple, accurate instrument for its measurement in 1972. Elevation of FEP in the erythrocytes first appears at 15-30 μg/100 ml in women and children and at 25 μg/100 ml in men (Roels 1976; Promelli 1972; Sassa 1973).

The health significance of the effects of low levels of lead in the blood on heme synthesis are not clearly known. A decrement in hemoglobin resulting in anemia first appears at blood lead levels of 40-50 μg/100 ml. (EPA 1977). This is slightly above the levels where an increase in ALA or FEP in the blood is seen. There is also some evidence that anemia may be caused in part by a shortened lifespan of erythrocytes caused by damage to the erythrocyte membrane. However, the inhibitory effect lead has on the synthesis of heme accounts for most of the decreased concentration of hemoglobin.

Because man appears to possess a large excess of ALAD, the health significance of ALAD inhibition remains doubtful (Roels 1976). The elevation of FEP probably has greater physiological significance; it is believed that a significant modification of FEP is not tolerable (Perlroth et al. 1966). It is possible that heme precursors are toxic in themselves. Elevated levels on ALA may have toxic effects on neuromuscular functions though this evidence was derived from animal studies using high doses of ALA (NAS 1980). The EPA (1977) concluded that elevation of FEP in the blood may also indicate impairment of mitochondrial function and cell respiration. Therefore, the elevation of FEP in the blood may be a good indicator of critical toxic effects of lead on the body.

C.   Effects on the Renal System

There is considerable information on the effects of lead to the kidneys. There are two different types of effects that can occur due to lead exposure. One is an acute form of renal tubular damage and the other is a chronic reduction in the ability of the kidneys to remove substances from the bloodstream. The former occurs with short term exposure and is reversible while the latter is considered to be of a slow, progressive nature.

The acute condition which results from lead poisoning is characterized by aminoaciduria (an excess of amino acids in the urine), glucosuria (glucose in the urine), and hyperphosphatura (an excess of phosphates in the urine). These symptoms reflect proximal tubular damages. (The tubules function is to send back into the blood chemicals that the body needs and leave waste products trapped outside.) Therefore, the lead interferes with the filtering process of the kidney which can deprive the body of nutrition. In a group of children with slight lead-related neurological signs, aminoaciduria was found in 8 of 43 children. Their blood lead levels ranged from 40 to 120 μg/100 ml (Pueschel et al. 1972). Chisholm (1968) found 20 of 23 children with chronic lead poisoning to have signs of aminoaciduria. There is evidence that acute lead induced renal effects can be successfully treated. When Chisholm treated the 20 children who showed signs of aminoaciduria, renal function in all returned to normal.

124

When an individual has been subject to prolonged exposure to lead, a different effect on the renal system can result. This disease is referred to as chronic lead nephropathy. It is characterized by slow development of the lead-exposed kidneys with arteriosclerotic changes, interstitial fibrosis, glomerular atrophy and degeneration of the vessels. All of these changes act to reduce the ability of the kidneys to remove substances from the blood stream. This disease can end in renal failure, even long after the exposure to lead is ended. Evidence of a causal relationship between chronic nephropathy and childhood lead poisoning was found in two studies that compared the deaths from chronic nephritis among people under 30 years of age in Queensland, Australia (WHO 1977). These cases involved childhood exposure with a latency of 10-30 years for the development of renal impairment. Chronic lead nephropathy is more common among people who have had a fairly high exposure to lead for more than 10 years than those exposed less than 10 years. This disease is difficult to detect in its early stages; in fact, individuals may lose up to two-thirds of the functional capacity of the kidneys and still be asymptomatic (EPA 1977).

In summary, acute renal effects can occur in children and adults with subtle signs of lead poisoning. Damage occurs in the proximal tubular cells resulting in decreased efficiency in filtering out proteins and other substances. This condition can be successfully treated. Prolonged exposure can lead to more serious effects on the renal system that are irreversible and may result in renal failure. Little is known about dose-response relationships for these effects.

D. Reproductive Effects

There is extensive evidence from occupationally exposed populations that lead,has serious effects on the human reproductive system. The effects include an increase in the number of miscarriages and stillborns, disruption of the ovarian cycle, loss of fertility in men, loss of libido and reduced potency in men and the increased likelihood of abnormal pregnancy.

Data on the adverse effects of lead on the reproductive system have existed since the turn of the century. A study done by Legge (1901) on 77 female lead workers found that out of 212 pregnancies, there were 90 miscarriages, 21 stillborns, and of the 101 live births, 40 infants died in the first year. The exposure to lead in this study was very high. However, a more recent study suggests that miscarriages occur in women with.only modest exposure (EPA 1979). The women is this study were wives of lead workers and not actually occupationally exposed to lead themselves. No actual levels of lead exposure were reported so no conclusions can be drawn about the minimally toxic level of exposure that would result in miscarriage.

It has also been found that subtoxic lead absorption during pregnancy is associated with preterm deliveries and premature fetal membrane rupture in term infants. Fahim et al. (1976) found the incidence of premature membrane rupture and preterm delivery to be 17 percent for an area 30 to 50 miles west of a lead mining area in Missouri and only .41 percent in a

Missouri urban area where no lead mining activities existed. The maternal and fetal blood lead levels at birth for the deliveries with preterm membrane rupture were 26 and 13 μg/100 ml, whereas for normal deliveries they were about 14 and 4 μg/100 ml respectively.

Lead can affect fertility as well as conception. Lancranjan et al. (1975) reported that moderately increased lead absorption (blood lead means = 52.8 μg/100 ml) resulted in decreased fertile ability in men. They found an increased number of malformed sperm, decreased number of total sperm, and sperm with decreased motility. They concluded that hypofertility induced by lead is probably due to lead's direct toxic effect on the gonads. Lead has also been associated with loss of libido and impotency but no dose-response relationship has been defined for these effects (EPA 1977). Lead also affects fertility in women. It has been estimated that short term exposure at ambient air levels of less than 7 μg/m^3 may cause an increase in anovular cycles (menstrual cycles in which ovulation does not occur) and disturbance in the lutein phase (ovulation occurs and the uterine walls in prepared for the egg to be implanted in this phase) (EPA, 1977).

Lead can pass through the placental barrier and accumulate in fetal tissues after about the 12th week of pregnancy. There is a high correlation between blood lead levels of women immediately after birth and the blood lead levels of their infants. Gershanik et al. (1974) found a correlation coefficient of .6377 for lead levels in infants and their corresponding mothers. There is little evidence to suggest that lead causes congenital malformations. However, since the neurobehavioral system of children is so susceptible to low exposures to lead, it is generally assumed that the fetus and newborn would be at least as susceptible. The study by Beattie (1975) (mentioned previously in the section on neurobehavioral effects) found an association between a high concentration of lead in the drinking water of pregnant women and mental retardation in their corresponding infants. Because there is so little evidence of the effects of prenatal lead exposure on mental functions in infants, it is impossible to define a dose-response relationship.

E. Carcinogenic Effects

The epidemiological evidence of the relationship between lead exposure and cancer is questionable. Several studies have been done to find the cause of death among people overly exposed to lead. Most of these studies found no significant correlation between lead exposure and deaths caused by cancer. However, there is evidence that lead causes cancer in laboratory animals.

A study done by Dingwall-Fordyce and Lane (1963) examined the causes of death among 267 men who died between 1926 and 1961. The men were classified as to lead exposure according to the nature of their work and if, they worked in an occupation with high lead exposure, according to their urinary lead excretion. They found in comparing the high lead exposure group to the low or negligible lead exposure groups that there was not a significant excess number of deaths caused by malignant tumors in the

126

high lead exposure group. There was an excess number of deaths but these were due mainly to vascular lesions of the central nervous system.

In another relevant study, beginning in 1938, orchardists who had sprayed fruit trees with lead arsenate at one time and the rest of the population living in the area of the spraying were classified into groups according to the degree of exposure to lead. In 1968, a follow up study was begun (Nelson et al. 1973). The status (living or dead) of the persons in the study was determined and if they had died the cause of death was determined. There was no suggestion of a relationship between lead exposure and death from three major causes of death: heart disease, cancer, and stroke.

In a more recent study (Cooper 1975) of mortality among lead smelter workers and lead battery workers, the causes of death that showed a statistically significant elevation included "all malignant neoplasms" and cancer of "other sites" in battery workers. However, the author concluded that the excess deaths due to neoplasms cannot be attributed to lead "because there was no consistent association between the incidence of cancer deaths and either length of employment or estimated exposures to lead".

There have been many studies done concerning the relationship of lead exposure and cancer mortality in laboratory animals. Several studies have shown that lead causes renal tumors in rats and hamsters, and one study each has associated lead with renal cancer in mice, lung tumors in hamsters and brain tumors in rats (EPA 1979). In all of these studies, the exposure to lead was very high, many times more than what a typical individual would be exposed to. Therefore, a dose-response relationship for much lower exposures to lead cannot be extrapolated from the data.

There is too little data to make a conclusion about the carcinogenicity of lead. If it is a carcinogen, then it is probably a weak one since the epidemiological studies done shown no significant association between lead exposure and cancer mortality. Further research is needed before any conclusions can be made.

F.   Effects on the Cardiovascular System

When exposed to a high level of lead over a prolonged period of time, arteriosclerotic changes (hardening of the arteries) can take place- in the kidney and lead to chronic renal disease (see section on renal effects). Dingwell-Fordyce and Lane (1963) reported a marked increase in mortality among lead workers due to cerebrovascular disease. This observation applied only to workers exposed to lead during the first quarter of the century, when lead exposure on the job was very bad. The same increase was not seen in workers employed more recently. In a more recent epidemiological study (Cooper 1975), there was no excess mortality due to diseases associated with hypertension or vasculopathy. It would appear from the studies done that lead affects the vascular system only with very high industrial exposure--like that seen around the turn of the century. These effects could either be direct effects on the blood vessels

themselves or a consequence of the toxic effects lead has on the renal system.

There is evidence that lead has toxic effects on the heart. Cases of structural and functional changes of the myocardiun (the muscular substance of the heart) have been described in adults and children with clinical lead poisoning (EPA 1977). In many cases when encephalopathy has been treated, the electrocardiographic abnormalities disappeared. Silver and Rodriguez-Torres found abnormal cardiograms in 21 of 30 children who had symptoms of lead toxicity. After therapy, the abnormalities were found in only 4 children.

Conclusive evidence of the effects of lead exposure on the cardiovascular system is not available so no dose-response relationships can be defined. The effects that have been noted may only affect those who have been exposed to a vary high level of lead.

G. Chromosomal Effects

The study of lead effects on chromosomes is technically difficult and therefore, it is hard to make any conclusions about these effects. The significance of the implication of injury to chromosomes is great. Each chromosome must separate correctly into two chromatids during cell division, and these chromatids must be equally redistributed in order to reproduce stable new cells for the maintenance of healthy tissue. Any chromosomal aberrations can be responsible for consequences as serious as defects in offspring of the affected individual (EPA 1977).

There are both negative and positive reports in this area. O'Riordan and Evans (1974) examined the lymphocytes of 70 male workers occupationally exposed to lead and found that chromosomal aberration frequencies were low and not significantly different from the control group. Bauchinger and Schmid (1976) also reported no increase in chromosomal aberration yield levels over the control group when they studied lymphocyte chromosomes of 20 industrial workers who showed a 20 to 30 percent increase in blood lead levels over the general population.

Studies done on 11 subjects (Forni et al. 1976) before and during initial occupation exposure to moderate quantities of lead fumes in a storage battery plant showed a doubling of the rate of abnormal cell divisions after one month. This rate increased after two months, remained in this range for seven months and then decreased somewhat. It must be noted here the small number of subjects studied making the results less reliable.

Chromosomal aberrations in lymphocytes of 24 workers in a zinc smelting plant who had increased blood levels of lead and cadmium were significantly higher than those of 15 members of the control group. Though it can't be determined which of the metals was to blame the authors suggest that the aberrations could be caused by the synergistic effects of several metal compounds (Bauchinger and Schmid 1972).

The evidence of chromosomal aberrations is conflicting and inconclusive. No clear dose-response relationship can be defined for the effects of lead on chromosomes. Because there have been studies showing an increase in chromosomal aberrations in lymphocytes, it may be that chromosomal damage occurs in other cells, possibly eggs and sperm (EPA 1977). The effects of lead on reproduction (notably miscarriages and stillborns) discussed previously may in part be due to the effects of lead on genetic material.

H. Gastrointestinal Effects

It has long been known that colic is associated with lead poisoning. Colic is usually an early symptom of poisoning.. It is seen most commonly in industrial exposure ccases but it is also a symptom of lead poisoning in infants and children (EPA 1977).

There is insufficient data by which to define a dose-response relationship for the effects of lead on the gastrointestinal system. One study of 64 men with blood lead levels between 40 and 80 $\mu g/100$ ml found 13 of those men to be suffering from colic and constipation (Beritic 1971). However, no conclusions as to the exposure required for colic to occur can be made from this study.

I. Effects on the Endocrine System

Effects of lead on the endocrine system are not well defined. It is known to decrease the thyroid function and possibly interfere with pituitary function (EPA 1977). There is not enough evidence in this area to make any conclusion about the effects or to define any dose-response relationship.

J. Effects on the Immunological System

Animal studies have indicated that exposure to lead may interfere with normal susceptibility to infection. Decrease production of antibodies may be one of the factors involved in this increased susceptibility. Williams et al. (1954) reported that lead binds antibodies in vitro and could do so in vivo. Again there is not enough evidence to make any conclusions about the effects of lead on the immunological system.

3. Conclusion

Though there is much known about the toxic effects of lead on the body, there is still controversy over what level of lead in the body should be considered safe. There is general consensus that toxic effects do occur in the body at minimum blood lead levels of 40 to 50 $\mu g/100$ ml. However, many researchers feel that a safe maximum blood lead level is closer to 25 $\mu g/100$ ml. Adverse neurobehavioral effects have been found to occur at blood lead levels of 20 to 25 $\mu g/100$ ml in children, and FEP elevation, which the EPA has concluded may indicate serious toxic effects, occurs at blood lead levels of 15 to 30 $\mu g/100$ ml. Therefore, the general population

may be subject to exposures to lead that produce deleterious effects. Whether this is the case may never be known since it would be virtually impossible to demonstrate these effects if the majority of the population were suffering from them.

The next chapter presents an empirical formulation of the theoretical model discussed in Chapter 3 using the St. Louis morbidity data set and the RAMS air pollution data. Specifically, the relationship between the presence of ozone and lead in the ambient air and the production of health is estimated. Other pollutants will also be examined for their effect on the production of health The results of this estimation allow benefits derived from a reduction in air pollution to be calculated.